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CALCIUM AND PHOSPHORUS METABOLISM IN DISEASES OF THE THYREOPARATHYREOID APPARATUS.

PART II.[1]

THE PROBLEM OF THE MODE OF ACTION OF VITAMIN D.

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Historical.

THE literature dealing with the function of vitamin *D* in the body, and the interrelationship of vitamin *D* and parathyreoid secretion is already extensive and covers a wide field of experimental study. Many factors influence the effect of vitamin *D*, for example, animal species, age of animal, composition of diet (especially as to calcium, phosphorus and nitrogen content) before and during the experimental study, the presence or absence of pathological conditions such as rickets or osteomalacia, the state of the parathyreoid apparatus, normal, hyperparathyreoidism, hypoparathyreoidism or aparathyreoidism *et cetera*. As the effect of vitamin *D* may vary with each of these other variables, it is apparent why experimental results have been discordant. No single theory has been expounded which satisfactorily covers all the observed phenomena. We intend to deal only with that aspect of the problem which is pertinent to the mode of action of vitamin *D* in overcoming the effects of parathyreoid insufficiency.

Soon after the preparation of irradiated ergosterol, Hess and Lewis,[62] from their experience in normal and rhachitic infants and from experimental work on a monkey, concluded that vitamin *D* acted by stimulating the parathyreoid glands. Stern[112]

[1] Part I of this paper was published in the issue of January 13, 1934.

was probably the first to publish results on the effect of irradiated ergosterol in overcoming the clinical symptoms of tetany. He gave "Vigantol" (dose not stated) to a patient suffering from chronic tetany, with great relief. Brougher from his results on thyreoparathyreoidectomized dogs[31] treated with irradiated ergosterol and a milk diet, and on five human beings suffering from hypoparathyreoidism[32] treated with irradiated ergosterol, concluded that "a rich supply of vitamin *D* is efficacious in enabling the body to utilize calcium in the disturbed calcium metabolism found in parathyroid tetany". In human beings he found a dosage as small as 4 to 20 drops of "Vigantol" (potency not stated) satisfactory, and states without giving figures that the serum calcium was raised.

Hansman[59] gave vitamin *D* in the form of "Radiostol" (one cubic centimetre = one milligramme irradiated ergosterol = 10,000 antirhachitic units) to six patients suffering from hypoparathyreoidism, and found the dosage necessary to insure adequate clinical improvement to vary between one and four cubic centimetres of "Radiostol" daily. This dosage was without appreciable effect on the serum calcium in the one case investigated. The action of vitamin *D* was considered to be synergic with the secretion of the parathyreoid glands.

Jones,[73] as early as 1926, found that large doses of cod liver oil (twenty cubic centimetres) if given daily for some days before thyreoparathyreoidectomy, prolonged life and prevented tetany. He found that if the animal had received vitamin *D* the serum calcium was of no guide as to the likelihood or not of actual tetany developing, and frequently found very low calcium figures without tetany in animals which had had cod liver oil before and after operation. This confirmed the observation of Steenbock, Jones and Hart[111] that the level of serum calcium did not always indicate the presence or absence of tetany, that is, that in the dog there was no tetanic level for calcium.

Greenwald and Gross[57] were unable to prevent tetany in dogs with cod liver oil or irradiated ergosterol, but of their two dogs treated with irradiated ergosterol one received it for only two days and the other for only three days. Cod liver oil did effect some improvement in several of their dogs, especially when calcium was also present in the food. They considered that the results of Jones,[73] Wade[127] and Brougher[31] were open to criticism in that there may have been parathyreoid tissue left, and that "the most likely cause of their beneficial results was that irradiated ergosterol stimulated parathyroid rests". These workers also confirmed the findings of Jones, and of Steenbock, Jones and Hart, that in animals that had had cod liver oil tetany may be absent with serum calcium figures below the accepted "tetanic level".

Taylor *et alii*[122] were able to relieve tetany by massive doses of irradiated ergosterol in dogs which had undergone extensive thyreoparathyreoidectomies. They did get some alleviation in dogs which had a more extensive dissection of the neck, but it was by no means so dramatic or complete, and they concluded from a very full discussion of a comparison of the results of parathormone injection and vitamin *D* administration, and of all the data as to the function of these two substances in various animals, that vitamin *D* acted by stimulating parathyreoid tissue which was not accessible to be removed at operation.

Morgan and Garrison[87] from experiments on dogs concluded that: "Vitamin *D* did not stimulate the production of parathyroid secretion, but intensified its effect." This somewhat general statement is not supported by the divergent effects of parathormone injection and vitamin *D* administration in rhachitic animals. ([96] for references and discussion.) Parathormone injections are found to intensify the rhachitic condition, while vitamin *D* leads to cure. Taylor *et alii* interpret the findings of Morgan and Garrison as indicating that parathormone and vitamin *D* stand in the relation of enzyme and co-enzyme.

In a series of very thoughtful papers Shelling[95] [96] [97] [98] [99] covers a very wide field of calcium and phosphorus metabolism, of the function of vitamin *D* and of the secretion of the parathyreoid glands and the relationship between each. He criticizes[96] the conclusion of Taylor *et alii*[122] by pointing out the great improbability that small (theoretical) remnants of parathyreoid tissue could in a few hours be stimulated to such activity by irradiated ergosterol that symptoms of gross hyperactivity result, and even death from hypersecretion. He points out that clinical hypoparathyreoidism, even in cases where only a single parathyreoid gland has been removed, is a very chronic disease that may remain stationary for many years. He considers that as spontaneous cure of hypoparathyreoidism is rare, the parathyreoid glands can have very little tendency to hypertrophy to meet physiological demands. Further, he points out that thyreoparathyreoidectomized animals whose condition has been alleviated by vitamin *D* administration relapse if the irradiated ergosterol is stopped. This has also been our experience clinically. The subjects in all the cases reported in 1930 have been kept under more or less constant observation. When from economic reasons they have been unable to procure their "Radiostol", they have rapidly drifted back to a hypoparathyreoidic state, and in one experimental case, E.A., we have definite information as to the time it may take for subjective symptoms and physical signs to reappear (see below) after cessation of vitamin *D* therapy. Shelling concludes that:

> Vitamin *D* in fairly large doses is capable of raising the serum calcium level and of ameliorating the symptoms of tetany independent of the parathyroid glands.

He also states that:

> With the data at hand no definite conclusions can be drawn as to the relationship of vitamin *D* and the parathyroids.

Jones, Rapaport and Hodes[74] concluded from their experimental results on thyreoparathyreoidectomized dogs that:

> The effects of vitamin *D* in alleviating tetany and keeping the animals alive, could hardly have been possibly due to a stimulation of the parathyroid glands.

Harris[60] states:

> Investigations on hypervitaminosis have substantiated the theory that the characteristic mode of action of vitamin *D* is to permit an increased "net absorption" of calcium and/or phosphorus from the gut, tending to raise the level of blood calcium and/or phosphate.

He goes on to show that this theory explains all the various phenomena and difficulties.

Kramer and Howland[76] state:

> Cod liver oil increases the availability of both calcium and phosphorus, and enables the body to function more economically with respect to these salts.

Bauer and his co-workers[19] from experiments on clinical cases of hypoparathyreoidism, concluded that:

> The beneficial results obtained in the treatment of parathyroid tetany when therapeutic doses of irradiated ergosterol are employed are directly related to the calcium intake and are not secondary to the stimulation of the parathyroid tissue.

There is therefore no unanimity as to the *modus operandi* of vitamin *D* and its relation to the parathyreoid glands in hypoparathyreoidism.

The Effect of Vitamin D Administration on the Level of Serum Calcium and Phosphorus and on the Excretion and Excretory Path of Calcium and Phosphorus.

Most of the literature deals with the effect of irradiated ergosterol in either human or experimental rickets, and very little work has been done on adult human beings. Kroetz[78] gave irradiated ergosterol to human beings above "therapeutic doses", and found a decrease in serum phosphorus and no change or a slight rise in serum calcium. [The term "therapeutic dose" of vitamin *D* is of no clinical or scientific value. In terms of cod liver oil 1,500 antirhachitic units (15 cubic centimetres of cod liver oil is the maximum British Pharmacopœia dose) is a large dose. In terms of irradiated ergosterol 60,000 units is well within the therapeutic range. Bauer *et alii*[17] consider 200,000 units a small dose.] Havard and Hoyle[61] gave irradiated ergosterol to adults in doses of four milligrammes (two subjects, one male, one female), and eight milligrammes (one subject, male) and found no variations of serum phosphorus or calcium above control periods. Unfortunately they make no mention as to whether the blood samples were taken fasting. This is especially significant as they state that they found a wide range of daily variations in both control and experimental periods. Rodecurt[92] found that vitamin *D* in the form of "Vigantol" raised the level of diffusible calcium, but as organic calcium salts were given by injection at the same time, the results are not very convincing.

The first systematic studies under adequate control conditions were carried out by Bauer, Marble and Claflin.[17] From experiments on normal subjects on a low calcium diet and on this diet supplemented by large doses of calcium chloride they concluded that doses of 5 to 20 milligrammes of irradiated ergosterol (50,000 to 200,000 antirhachitic units) caused effects which were slight and not constant and probably not significant. On 30 milligrammes of irradiated ergosterol they found a fall in fæcal calcium and phosphorus, and a rise in urinary calcium and phosphorus, the calcium content of the diet making no difference. They considered that "the results indicated an increased absorption from the intestine and increased excretion in the urine without any retention". There was a slight rise in serum calcium on all dosages, and a slight rise in serum phosphorus on the higher dosage. Following the cessation of irradiated ergosterol the fæcal calcium and phosphorus rose promptly and the urinary calcium and phosphorus fell slightly.

Bauer and Marble[8] found in a case of osteoporosis and in a case of osteomalacia with tetany due to faulty absorption from the intestine, that the administration of irradiated ergosterol led to striking clinical improvement. The changes in calcium and phosphorus metabolism were of the same type as in normal individuals, but the dose of irradiated ergosterol necessary to produce these changes was smaller (eight to ten milligrammes *per diem*). The rise in serum calcium and phosphorus was more marked. In the case of osteomalacia there was a large retention of calcium and phosphorus. Following the scheme developed by Albright, Bauer, Cockrill and Ellsworth[2] for classifying the disorders of calcium and phosphorus metabolism, Bauer and Marble consider that the reason why irradiated ergosterol has such little effect in normal individuals is that as the body stores are saturated, there is no further need for calcium, so it is excreted; while in calcium deficiency diseases, such as rickets and osteomalacia, the body stores are not saturated and irradiated ergosterol overcomes the failure to absorb sufficient calcium and phosphorus from the gastro-intestinal tract, and this calcium and phosphorus may then be utilized for bone formation.

In a fourth paper from the same laboratories, Bauer, Marble and Claflin[19] give their findings in metabolic studies on the effects of irradiated ergosterol administration in hypoparathyreoidism, and conclude:

> 1. The beneficial results obtained in the treatment of parathyroid tetany when therapeutic doses of irradiated ergosterol are employed are directly related to the calcium intake and are not secondary to the stimulation of remaining parathyroid tissue.
>
> 2. The administration of five milligrammes of irradiated ergosterol a day to an individual with parathyroid tetany on an inadequate calcium intake is without beneficial effects.
>
> 3. The administration of five milligrammes of irradiated ergosterol a day to an individual with parathyroid tetany receiving a high calcium intake produces the following changes: (*a*) A decreased fæcal calcium excretion. (*b*) An increased urinary calcium excretion. (*c*) An increased positive calcium balance. (*d*) A rise in the serum calcium. (*e*) An increased fæcal phosphorus excretion. (*f*) An increased urinary phosphorus excretion. (*g*) A decrease

in the positive phosphorus balance. (*h*) A fall in the serum phosphorus. (*i*) The serum calcium rises faster than the serum phosphorus falls, and therefore the Ca × P product rises. (*j*) The signs and symptoms of tetany disappear.

4. The dose of irradiated ergosterol required to produce beneficial changes in the calcium and phosphorus metabolism of individuals with calcium and phosphorus disorders is smaller than the amount necessary to produce similar changes in the calcium and phosphorus metabolism of normal individuals.

Conclusion 3 (*i*) arises from their discussion on the classification of Albright, Bauer, Cockrill and Ellsworth, when they state:

If the changes in the calcium and phosphorus metabolism observed in these patients are interpreted as being secondary to increased calcium absorption from the gastro-intestinal tract, they can be readily explained on the basis of the classification of disorders of calcium and phosphorus metabolism given by Albright, Bauer, Cockrill and Ellsworth.[29] According to this classification, disorders of the parathyroid glands represent conditions in which the body fluids contain the saturating amount of calcium phosphate, but the proportion of the calcium ion to the phosphate ion is abnormal. In parathyroid tetany the calcium ion is reduced and the phosphate ion is increased. Because the body fluids already contain a saturating amount of calcium phosphate, the rise in the serum calcium (due to the increased absorption of calcium from the gastro-intestinal tract) is accompanied by a fall in the serum phosphorus and an increased phosphorus excretion in an attempt to keep the Ca × P product normal. However, the fall in the serum phosphorus is not sufficiently rapid and therefore the Ca × P product rises.

They remark on the effect of irradiated ergosterol in "overdosage", and consider that this action of irradiated ergosterol is probably due to contained impurities.

It has been necessary to give the findings of these workers in some detail because they have a special clinical significance and because their conclusions cannot be accepted on the evidence available. They consider that the ability of irradiated ergosterol to raise the level of serum calcium and phosphorus is dependent on the calcium and phosphorus intake. Shelling,[96] by giving large doses of irradiated ergosterol on a calcium-free diet, was able to raise the level of serum calcium in completely parathyreoidectomized rats to normal and even hypercalcæmic levels. This cannot be explained as due to the presence of "toxic" substances in the irradiated ergosterol, because if the diet contained very small amounts of calcium, 0·012% of the diet, he could bring about similar changes in the serum and relief from tetany on comparatively small doses of irradiated ergosterol. It seems as if the calcium in the intestines is "available" to be acted upon by irradiated ergosterol, but that calcium once deposited in the bone is no longer available to be acted upon by irradiated ergosterol until the concentration of irradiated ergosterol has reached a high level.

Bauer and his co-workers tacitly assume that the fæcal calcium is unabsorbed calcium, and a decrease in fæcal calcium on irradiated ergosterol administration means better absorption. However, it is well known that the bowel is the chief excretory path of calcium, and it may well be that the same amounts of calcium are absorbed in hypoparathyreoidism with or without irradiated ergosterol, but that with irradiated ergosterol less is excreted. This aspect has recently been stressed by Clouse[36] and by Harris.[60]

It is difficult to understand why the greater absorption or lessened excretion of calcium in hypoparathyreoidism can *per se* lead to an alleviation of symptoms, as the arguments of Bauer *et alii* make out; normally hypoparathyreoidism is associated with a positive calcium balance[53][121][7] as is the metabolism after removal of parathyreoid tumours;[4][69][70][129] why then should merely making the balance more positive prevent the onset of tetany?

Bauer *et alii* consider that the rise in serum calcium with administration of irradiated ergosterol is secondary to a greater absorption of calcium. Now they admit that prior to giving irradiated ergosterol the serum is saturated with calcium phosphate, but on giving irradiated ergosterol the Ca × P product increases and it seems more logical to consider that the primary change caused by irradiated ergosterol is in the capacity of the serum to hold a greater Ca × P product, and that the absorption or lessened excretion of calcium is secondary. They argue that irradiated ergosterol has more effect in osteomalacia and osteoporosis than in normal individuals, because in the latter the body stores of calcium are saturated. But if this is logical, then why should it have more effect (as it certainly does) in hypoparathyreoidism than in normal people? It is a well-known fact that there is no deficiency in calcification in hypoparathyreoidism.

Experimental Data.

In Part I of this paper we presented experimental data for the calcium, phosphorus and nitrogen balances on two patients suffering from hyperthyreoidism with an associated hypoparathyreoidism. We now wish to discuss certain other data obtained from these two patients during the period of vitamin *D* administration.

Data from Edith A.

In the first place we shall discuss data obtained from Edith A.

Clinical Changes Induced by Vitamin D Administration.—Within the first few days there was a marked symptomatic improvement which was continued throughout the whole period of vitamin *D* administration and for approximately twenty-six days after its cessation, by which time both the signs and symptoms of hypoparathyreoidism had returned. (See case report.) Though there was no subjective change when the dose of irradiated ergosterol was increased from three to six milligrammes or from six to nine milligrammes *per diem*, the medical and nursing staff both thought she was not so well on the larger doses. From our general experience with irradiated ergosterol therapy we consider that the smallest dose which insures freedom from subjective symptoms and from cramps

either remains adequate or can gradually be reduced. We have not had occasion to increase a dose that has been sufficient. There is some indication that the required dose is less in summer than in winter.

Changes in Serum Calcium (see Table X and Graph XI).—The serum calcium rose from 9·7 milligrammes *per centum* to 10·5 milligrammes *per centum* within the first week of vitamin *D* administration, then fell within the next week to 10 milligrammes *per centum* and remained at this level during the remaining fortnight that three milligrammes of irradiated ergosterol were given. The serum calcium rose to 10·7 milligrammes *per centum* when the dose of irradiated ergosterol was increased to six milligrammes daily, but did not rise further when nine milligrammes of irradiated ergosterol were given daily. When the administration of vitamin *D* ceased the serum calcium gradually fell, and by the twenty-sixth day had reached a level of 9·3 milligrammes *per centum*.

Changes in Serum Inorganic Phosphorus.—The inorganic phosphorus showed changes in an opposite direction, but of relatively greater amount than those of the serum calcium. The full effect of vitamin *D* on the serum phosphorus lagged behind that of the serum calcium. Starting at a level of 5·7 milligrammes *per centum*, the phosphorus fell to 5·5 milligrammes *per centum* in seven days, then to 4·7 milligrammes *per centum* in a further seven days, and remained at this level till the second period of the administration of nine milligrammes of irradiated ergosterol daily, when it fell to four milligrammes *per centum*. It may be that this further fall represents a lag effect of the administration of six milligrammes of irradiated ergosterol daily, and would have still occurred had the dose of six milligrammes been continued for a further two periods. Twenty-six days after the cessation of vitamin *D* administration the inorganic phosphorus had reached a level of 5·1 milligrammes *per centum*.

Changes in Calcium and Phosphorus Balance During Vitamin D Administration.—These changes have been discussed in Part I. There was an increased storage of both calcium and phosphorus, over that of the fore-period. The increased retention of phosphorus is in contrast to the findings of Bauer *et alii*.[19] These workers found an increased excretion of phosphorus in both stool and urine. This difference in result is probably due to the difference in experimental conditions, especially as regards the difference in the calcium intake.

Changes in Neuro-Muscular Response to Electrical Stimulation During the Administration of Vitamin D.—Before the administration of vitamin *D* there was a characteristic tetanic response to alternating current and an exaggerated response to the kathodic closing contraction of the galvanic current. During the administration of irradiated ergosterol the response to galvanic current was tested at weekly intervals. After the cessation of vitamin *D* it was tested more frequently at first, and then weekly. For the first few tests we had no means of measuring the exact amperage, but as the responses were always tested by the same competent observer the results are of comparative value. From the table (Table D) it will be seen that while vitamin *D* had a very definite effect in lowering the excitability of the neuro-muscular mechanism, the effect was not proportional to the actual quantity of irradiated ergosterol administered; doses of six to nine milligrammes of irradiated ergosterol had no greater effect than three milligrammes. After the cessation of vitamin *D* there was a gradual change in the response to the galvanic current, but as the nature of the test does not allow of variations of less than a milliampère being measured, there is no definite record of an increased excitability till the twenty-sixth day, by which time all the muscle groups tested reacted to a milliampère less than that required during vitamin *D* administration. Though it was instructive to follow the response to galvanic current in this case, our experience of testing neuro-muscular responses in normal and hyperthyreoidic subjects leads us to doubt its utility as a means of investigation.

Data From Emma B.

In the second place we shall consider data from Emma B.

Clinical Changes Induced by Vitamin D Administration.—Though feeling fairly well on admission to hospital, this subject experienced marked subjective improvement on two milligrammes of irradiated ergosterol, and at the end of two four-day periods she felt so well she would not stay in hospital any longer.

Changes in Serum Calcium and Phosphorus (see protocols).—The level of serum calcium was uninfluenced by vitamin *D* administration. The serum inorganic phosphorus after a lag fell slightly. The absence of change in the level of serum calcium and phosphorus illustrates that symptomatic improvement is independent of the actual level of serum calcium or phosphorus.

Changes in Calcium and Phosphorus Balance.—There was very little change in calcium balance, and there was, as in Edith A., slightly increased positive phosphorus balance.

The metabolic changes in this patient confirm the findings in the previous patient.

Discussion.

The Relationship Between Symptomatic Improvement and Changes in the Level of Serum Calcium and Phosphorus During the Administration of Vitamin D.

It is difficult to believe that there is a direct relationship between the marked symptomatic improvement and the slight changes in concentration of serum calcium and phosphorus in hypoparathyreoidic patients treated with irradiated ergosterol.

Bauer, Marble and Claflin[19] do not state whether their hypoparathyreoidic patient (Case III) improved clinically while taking irradiated ergosterol on a low calcium diet; at this time the serum

calcium was unchanged. As discussed earlier in this paper, experimental hypoparathyreoidic animals have shown marked improvement without changes in serum calcium levels when given vitamin *D*. Our subject, Edith A., experienced marked symptomatic improvement within a few days of the commencement of vitamin *D* administration, and though at the end of a week the serum calcium had risen from 9·7 milligrammes *per centum* to 10·5 milligrammes *per centum*, the phosphorus level was practically unchanged. At the end of a fortnight the clinical improvement was maintained, though the serum calcium had fallen to 10 milligrammes *per centum*, the serum phosphorus being 4·7 milligrammes *per centum;* and when with a dose of six milligrammes of irradiated ergosterol daily the serum calcium had risen to 10·7 milligrammes *per centum*, there was no improvement in signs or symptoms over that shown during the administration of three milligrammes of irradiated ergosterol daily. Similarly in our subject, Emma B., there was definite symptomatic improvement, though the serum calcium and phosphorus remained constant. Likewise in a patient, A.H., reported by Hansman[59] there was a very marked clinical improvement without any change in the level of serum calcium. This patient, who has been constantly under observation since 1929, had no irradiated ergosterol for three weeks between January 10, 1933, and January 31, 1933; on the latter date her serum calcium was 9·0 milligrammes *per centum* and serum phosphorus 5·7 milligrammes *per centum*. She was experiencing cramps and numbness, and was very depressed and irritable. She was instructed to take the smallest dose of "Ostelin" daily that would just relieve her symptoms. Twenty drops daily was found adequate. At the end of two weeks her serum calcium was 10 milligrammes *per centum* and phosphorus 5·8 milligrammes *per centum*. Four weeks later, on the same dose, her serum calcium was 9·4 milligrammes *per centum*. She felt very well, was not irritable or depressed, and had no cramps or numbness.

The clinical improvement is likewise independent of the Ca × P product. Though the actual concentration of calcium does not change, it is still quite possible that there may be important changes in the relative amounts of ionized, diffusible and organic calcium. Research to determine this possibility is needed.

The Relationship Between the Symptomatic Improvement and Changes in Calcium and Phosphorus Balance.

Bauer *et alii*[19] concluded that the improvement in signs and symptoms of hypoparathyreoidism which follows the administration of vitamin *D* is a consequence of the better absorption of calcium. In our patient, Edith A., the calcium intake was 0·7 gramme *per diem*. In the fore-period there was an average negative calcium balance of −0·029 gramme *per diem*, which was no greater than in the normal control, Joan H., on an intake of 0·44 gramme *per diem*, so that failure of absorption could hardly have been of much moment. During vitamin *D* administration there was a positive balance of 0·09 gramme *per diem*, so that according to Bauer, an additional absorption of 0·12 gramme in a total of 0·7 gramme *per diem* would be sufficient to account for the marked clinical improvement. If the conclusions of the Boston School on calcium metabolism in hyperthyreoidism and in hypoparathyreoidism were both correct, we should have in our cases of hyperthyreoidism associated with hypoparathyreoidism the hyperthyreoidism causing a gross excretion of calcium in the fæces and the hypoparathyreoidism preventing adequate absorption of calcium; but instead of a large calcium loss these patients were in equilibrium. We consider that the slightly better calcium balance which accompanied vitamin *D* administration cannot be considered the direct cause of the symptomatic improvement. It is quite possible that on smaller doses of irradiated ergosterol there would have been the same clinical improvement without any measurable change in calcium and phosphorus balance over the fore-period.

It seems to us that apart from any function vitamin *D* has in raising the serum calcium and effecting changes in serum phosphorus, and in modifying the absorption and excretion of calcium and phosphorus, its chief function over a very wide range of concentration is in making calcium available for tissue metabolism. It changes the tissues or the circulating calcium in some way; the amount required to induce this change varies inversely with the level of diffusible serum calcium. With normal parathyreoid function sufficient vitamin *D* is obtained from the quantities present in food or produced by solar radiation from precursors in the skin. In hypoparathyreoidism larger amounts are necessary because the amount of diffusible serum calcium is greatly decreased.[58] Similarly, the amount of vitamin *D* required varies with the calcium and phosphorus intake; when these elements are available in adequate amounts and optimal proportions, the amount of vitamin *D* required is small; when there is a low calcium intake or a high phosphorus : calcium ratio, more vitamin *D* is required, not to determine the absorption of calcium, but to insure its utilization. In other words, we postulate that vitamin *D* is a catalyst accelerating intimate chemical changes by means of which inorganic calcium becomes a physiological component of tissue. We have not made a comprehensive survey of the literature on rickets to see whether this theory has been previously brought forward, but no mention of such is given in the four last surveys of our knowledge of the mode of action of vitamin *D*.[34][36][60][126] However, Kramer and Howland's idea as to the mode of action of vitamin *D* might be interpreted as expressing the same general idea.

Conclusions.

1. There is no unanimity as to the *modus operandi* of vitamin *D* and its relation to the parathyreoid glands in hypoparathyreoidism.

2. The beneficial effect of vitamin *D* in hypoparathyreoidism is not due to better absorption of calcium from the intestinal tract, to changes in the serum level of calcium, inorganic phosphorus or the Ca × P product.

3. We suggest that vitamin *D* acts by making calcium available for tissue metabolism.

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APPENDIX.

Case Histories.

The following are the case histories of the patients referred to in the paper:

ALBERT I., a male, married, aged thirty-eight years, was admitted to hospital under care of Dr. C. G. McDonald. He had complained of symptoms for one year. He was shaky, "nervy", and had lost weight (about 12·7 kilograms) in spite of a good appetite. He had palpitation and shortness of breath on exertion, and a subjective feeling of heat. Perspiration had not increased. He had a swelling in the neck for five months. He had weakness and huskiness of voice. He had appendicectomy eighteen months ago.

Physical examination revealed an apprehensive facies, with classical appearance of hyperthyreoidism. Speech was jerky and hurried. He was very fidgety and unsteady. His hands were warm and moist, had marked tremor and trophic changes. Marked exophthalmos was present, the corneæ were glistening, the eyes were staring; a definite von Graefe sign was present. Marked enlargement of the whole thyreoid gland was present. It was uniformly soft and no nodules were palpable; it was very vascular, and a marked bruit was audible all over the gland. The patient had had no treatment prior to admission.

On April 29, 1932 (as an out-patient), the basal metabolic rate was +51%, pulse 90 per minute; weight 51·4 kilograms.

On May 17, 1932, he was admitted to the metabolic ward, after being in a general ward for several days. His basal metabolic rate was +29%; basal pulse rate 104 per minute; weight 50 kilograms. The experimental diet was now begun.

On May 23, 1932, the first experimental period commenced.

On June 3, 1932, the basal metabolic rate was +36%; the basal pulse rate 116 per minute; the weight was 50·7 kilograms. Treatment, consisting of Lugol's iodine, 3 cubic centimetres daily, was begun and continued for four periods.

On June 17, 1932, the basal metabolic rate was +9·3%.

On June 21, 1932, a subtotal thyreoidectomy was performed by Mr. Poate.

The pathological report from Dr. G. Davies on the gland was as follows: Macroscopic: The right lobe and half the left lobe of a much enlarged thyreoid gland. Weight 108·5 grammes. On section the tissue has a pale amber-grey colour and colloid is not obvious on macroscopic examination. Microscopic: Considerable diffuse hyperplasia is present. Alveoli are mainly small, and are lined by large cuboidal and low columnar cells. The colloid is not pale, but peripheral vacuolation is a marked feature. In some alveoli only a trace of colloid remains—others are empty. Many lacunar alveoli are present. There is considerable interlobular fibrosis, but almost no lymphoid accumulation. Diagnosis: Diffuse hyperplastic goître.

On July 14, 1932, the patient returned to the experimental ward, and had three further experimental periods, the first of which was not used.

Comment.—This is a classical case of hyperthyreoidism.

DOROTHY E., a female, single, aged twenty-two years, was admitted to hospital from Dr. R. Flynn's out-patient clinic. She was well up to three months ago. She then began to have breathlessness, but no palpitation. She had a subjective feeling of heat; was "nervy"; had lost weight in spite of a good appetite; was fidgety and easily upset. Her periods were regular and unchanged. She had headaches over the whole of her head. She had noticed a swelling in the neck for three months; had had difficulty in swallowing, choking feeling, and phlegm in back of throat for three months.

Physical examination revealed a somewhat unhealthy appearance. The hands were warm and moist; a definite tremor was present with hyperextension. The eyes showed definite exophthalmos and a glistening cornea; palpebral fissures were widened; von Graefe's sign was present. The thyreoid was moderately enlarged, smooth, regular, contained no nodules, was soft and vascular; and a bruit was heard over whole of the right side.

The previous history contained nothing of importance. The patient had had no previous treatment before admission to the metabolic ward.

On October 20, 1932, the patient was admitted to the metabolic ward, and the experimental diet was commenced.

On October 21, 1932, the basal metabolic rate was +75%; the basal pulse rate was 104 per minute; the weight was 51·8 kilograms.

On October 22, 1932, the first experimental period was begun.

On November 3, 1932, the basal metabolic rate was +52%; the basal pulse rate was 112 per minute; the weight was 51·5 kilograms. Lugol's iodine 1·8 cubic centimetres *per diem* was administered and continued till the end of the study. During the course of study, on December 5, 1932, the patient had right-sided abdominal pain and a temperature of 37·2° C. for one day, and on December 13, 1932, one tooth was extracted.

Comment.—This is a classical uncomplicated case of hyperthyreoidism.

DARCIA T., aged twenty-one, a female, single, was on February 27, 1931, referred for estimation of basal metabolism from the Royal Hospital for Women, Sydney, where she was a trainee.

She complained of dyspnœa on exertion for twelve months; six months ago she was off duty for three months on account of œdema of the legs, which was considered to be either of renal or cardiac origin; no definite diagnosis was made. On returning to duty she still had dyspnœa on exertion and palpitation. She complained of being irritable and nervy. She had slight loss of weight in spite of a good appetite. She had subjective feeling of heat and

increased perspiration. She did not know she had a swelling in the neck.

Physical examination revealed an unhealthy looking girl, fidgety. Speech was hurried. Her hands were warm and moist; tremor and trophic changes were present. The eyes showed slight exophthalmos, the left eye being more marked than the right; there was a von Graefe sign. In the thyreoid moderate enlargement affected the whole gland uniformly, no nodules were palpable; the gland was soft to palpation and was vascular; a bruit was present at both lower poles. The basal metabolic rate was +40%; the basal pulse rate was 100 per minute; the weight was 51·4 kilograms.

On September 4, 1931, the patient again reported for estimation of basal metabolic rate. She had had no treatment in the *interim*, and had finished her training. Physical examination was practically that of February 27, 1931. The basal metabolic rate was +36%; the basal pulse rate was 96 per minute; the weight was 54·5 kilograms.

On September 7, 1931, she was admitted to the metabolic ward for study.

On September 19, 1931, the experimental diet was commenced.

On September 22, 1931, the first experimental period began.

On October 2, 1931, the basal metabolic rate was +20%; the basal pulse rate was 100 per minute; the weight was 53·5 kilograms. Lugol's iodine, 1·6 cubic centimetres daily, was commenced.

On October 5, 1931, the basal metabolic rate was +13·5%; the basal pulse rate was 100 per minute; the weight was 53·6 kilograms. Lugol's iodine, 2 cubic centimetres daily, was given.

On October 9, 1931, the basal metabolic rate was −2·5%; the basal pulse rate was 94 per minute; the weight was 53·6 kilograms. The thyreoid was very firm. A bruit was heard over the right lower lobe.

On October 27, 1931, the basal metabolic rate was +6%; the basal pulse rate was 90 per minute; the weight was 56·1 kilograms. Iodine administration was suspended.

On November 10, 1931, an exposure to superficial X rays was given. The basal metabolic rate was +30%; the basal pulse rate was 108 per minute; the weight was 55·6 kilograms. The thyreoid was larger and softer; it was more vascular and a bruit was audible over the right lobe.

On November 20, 1931, the basal metabolic rate was +28%; the basal pulse rate was 118 per minute; the weight was 55·0 kilograms. The patient was not feeling so well. She had headaches and felt nauseated. She had lost weight. Speech was hurried. She was fidgety. Definite tremor and hyperextension were noted. The thyreoid was firmer and smaller; a bruit was still present.

On December 1, 1931, the basal metabolic rate was +43%; the basal pulse rate was 118 per minute; the weight was 55·6 kilograms.

On December 2, 1931, a second exposure to X rays was given.

On December 15, 1931, the basal metabolic rate was +27%; the basal pulse rate was 108 per minute; the weight was 52·8 kilograms. The study was terminated.

MARJORIE R., a female, aged twelve years, was on May 29, 1932, admitted to hospital under care of Dr. L. Hughes.

The patient was an inmate of a state home, and had been on diet of low calcium content. Intelligence was poor, and the history of illness was not satisfactory. The illness apparently was of recent origin. No definite symptoms could be elicited. The matron of the home said that the patient had an enormous appetite and would eat anything the other girls left. She was reported to be irritable and lazy. The patient admitted she became tired easily and had shortness of breath, but no palpitation. She had always had nocturnal enuresis.

Physical examination revealed an extremely wasted child. The face and eyes were suffused. She was fidgety. She had a typical appearance of hyperthyreoidism. The hands, which were markedly hyperextended, were warm and moist; definite trophic changes were present. The eyes showed definite exophthalmos, widened palpebral fissures and a definite von Graefe sign. The thyreoid was definitely enlarged in both lobes, was firm to palpation and vascular; and on auscultation a bruit was heard all over the gland. Definite tachycardia was present.

Treatment previous to admission had consisted of Lugol's iodine, 0·36 cubic centimetre (six minims) daily for one month, but no iodine had been received during the last four weeks.

On May 20, 1932 (as an out-patient), the calories per square metre per hour were estimated at 55; the basal pulse rate was 156 per minute; the weight was 30 kilograms.

On June 3, 1932 (as an in-patient), the calories per square metre per hour were 51; the basal pulse rate was 132 per minute; the weight was 30 kilograms.

At the end of a fore-period of 36 days she was given X ray therapy, two exposures, and then a period of twelve days on 1·8 cubic centimetres (thirty minims) of Lugol's iodine.

She left hospital to go to an orphan's home, and rested in bed in the morning. While in the home she had two exposures to X rays. The hyperthyreoidism progressed and she had to be readmitted to hospital. Her general condition grew worse, the thyreoid enlarged markedly, and she continued to have tachycardia. The calories per square metre per hour rose to a maximum of 55·8, and thyreoidectomy was decided upon, but up to the time this paper went to the press she could not be brought to a satisfactory state for operation.

PHYLLIS W., aged twenty, a female, single, was admitted to hospital under the care of Dr. S. A. Smith. She gave a history of swelling in the neck and symptoms of hyperthyreoidism for two years. Lately symptoms had been getting worse. She complained chiefly of being "nervy" and fidgety, and of the swelling in the neck. She had no palpitation, but complained of shortness of breath. She had subjective feelings of heat and increased perspiration. She was losing weight, but had been eating in order to avert the loss. Her appetite was good and her bowels moved regularly. Menstruation was regular and had not changed. She suffered from frontal headaches since the onset of hyperthyreoidism. She was not conscious of phlegm in the throat.

Physical examination revealed an unusually stolid type of patient to have hyperthyreoidism. Mentality was poor. Her hands felt warm and moist; no tremor and no trophic changes were present. The eyes showed marked exophthalmos, and all the classical eye signs were also marked. The thyreoid was palpable on both sides, but the right side was larger than the left; the tumour was soft and the outline difficult to make out; there was a marked bruit audible over the right lower lobe. The heart was rapid and regular, but the pulse was often very soft.

Previous treatment: She had been treated with iodine for the first twelve months of her illness, but had had no iodine for a year.

On January 6, 1933, the basal metabolic rate was +50%; the basal pulse rate was 126 per minute; the weight was 56 kilograms. The experimental diet was begun. Rest in bed was ordered, but no other medication was given.

On January 10, 1933, the first experimental period commenced.

On January 20, 1933, the basal metabolic rate was +30%; the basal pulse rate was 112 per minute; the weight was 55·2 kilograms. The first exposure to superficial X rays was given.

On February 10, 1933, the basal metabolic rate was +36%; the basal pulse rate was 124 per minute; the weight was 55·2 kilograms. Treatment consisted in a second exposure to superficial X rays.

On March 7, 1933, the basal metabolic rate was +30%; the basal pulse rate was 104 per minute; the weight was 56 kilograms. A third exposure to superficial X rays was given.

During the course of the experimental study she had teeth extracted on several occasions without any noticeable effect on her general condition. The general condition showed very little change. The thyreoid gland grew a little smaller if anything, but there was no definite change.

Comment.—This was a case of moderately severe chronic hyperthyreoidism.

DULCIE S., aged seventeen, a female, married, was admitted to hospital under the care of Dr. E. W. Fairfax on January 2, 1933. She sought medical attention because she had been vomiting for a week and had a stye and a sore nose. She had to be questioned before she admitted to any other symptoms. The mother had noticed a swelling in the neck for a year. She had been "nervy" and easily upset, fidgety and irritable for six months. She had had shortness of breath and palpitation for three months. She had always had headaches and they had not changed. She had always felt the heat, and had not been affected by the present summer any more than usual. Her appetite was large. Her weight varied; she used to be 38·2 kilograms, on admission her weight was 34·5 kilograms. Her bowels moved regularly. She had no phlegm in the back of the throat. Her voice used to get husky. She was married when fifteen years old and had a child twenty months old, which she had suckled till her admission to hospital. Menstruation returned a few days before admission to hospital.

Physical examination revealed a very diminutive person in every sense. She was fidgety. Her speech was not hurried, but her voice was weak and husky. Her hands were unsteady with definite hyperextension; no definite trophic changes were present. The eyes showed all the classical signs. There was definite tachycardia. Definite uniform enlargement of the whole thyreoid gland was present; there were no palpable nodules; consistency was moderately firm; it was vascular to palpation; a bruit was audible over the whole gland.

On January 8, 1933, she was transferred to the metabolic ward. The experimental diet was commenced.

On January 10, 1933, the basal metabolic rate was +59%; the basal pulse rate was 120 per minute; the weight was 34·5 kilograms. The experimental study began. Treatment consisted in rest in bed, no medication was used.

On January 20, 1933, the basal metabolic rate was +61·5%; the basal pulse rate was 128 per minute; the weight was 34·3 kilograms. The condition was stationary. An exposure to therapeutic superficial X rays was given.

On January 26, 1933, as the patient was slowly losing weight, the diet was increased.

On February 10, 1933, the basal metabolic rate was +56%; the basal pulse rate was 128 per minute; the weight was 33·6 kilograms. An exposure to therapeutic deep X rays was given.

On February 14, 1933, the basal metabolic rate was +56%; the basal pulse rate was 128 per minute; the weight was 33·6 kilograms. The diet was further increased.

On March 3, 1933, the basal metabolic rate was +35%; the basal pulse rate was 126 per minute; the weight was 34·5 kilograms. Lugol's iodine, 1·8 cubic centimetres, was given daily for twenty-four days.

On March 30, 1933, a subtotal thyreoidectomy was performed by Sir John McKelvey.

Comment.—This was a case of severe hyperthyreoidism in an adolescent married female in which we attempted to influence calcium balance by X ray therapy and with iodine therapy without effect. The family history of this patient was interesting. Her maternal grandmother died, aged fifty-two years, of cerebral hæmorrhage. This good lady was a great-grandmother at fifty years of age, which must be a somewhat unique occurrence amongst people of British stock.

JEAN MCL., a female, single, aged twenty-three years, was admitted to hospital under care of Dr. B. Edye on November 9, 1932, complaining of breathlessness, palpitation of heart and swelling of feet for the last six months. She had noticed a swelling in the neck for the same length of time; she was not "nervy", but was fidgety. She had lost a little weight, though her appetite was good. Her bowels moved regularly. Her menstrual cycle was regular, but the flow had become less copious. She had subjective feelings of heat and had always felt hot for the last six months. She had temporal headaches lately. She had phlegm in the back of her throat.

Physical examination revealed a healthy-looking girl, somewhat fidgety. Her speech was not hurried, but her voice was weak and husky. Her hands were warm and moist; there was hyperextension but no tremor. The eyes showed slight exophthalmos, the palpebral fissures were widened, the cornea was glistening and there was a definite von Graefe sign. The thyreoid gland was definitely enlarged; both lobes were equally affected, the contour was regular and no nodules were palpable. The gland was very vascular to palpation, and a thrill was present over both upper poles. A typical bruit was audible over the whole gland. There was definite tachycardia.

On November 11, 1932, the patient was transferred to the metabolic ward. The basal metabolic rate was +57%; the basal pulse rate was 108 per minute; the weight was 51·7 kilograms. The experimental diet was begun.

November 15, 1932, was the first day of the fore-period. Rest in bed was ordered, but no medication.

On November 29, 1932, the basal metabolic rate was +38%; the basal pulse rate was 88 per minute; the weight was 50·7 kilograms. Deep X ray therapy was commenced and continued till December 14, 1932. The patient had eight exposures.

On December 20, 1932, the study was terminated. This patient left hospital but did not improve. She was readmitted on February 21, 1933, to a general ward.

On February 22, 1933, the basal metabolic rate was +36%; the basal pulse rate was 120 per minute; the weight was 55·9 kilograms.

On March 1, 1933, a subtotal thyreoidectomy was performed. The histological report by Dr. G. Davies was as follows: Macroscopic: The right lobe, part of the left lobe and a large pyramidal lobe of an enlarged thyreoid gland. Weight 69 grammes. On bisection the tissue is seen to have a very pale pink colour. No amber colour is seen. Microscopic: Very marked and widespread hyperplasia is present. Almost without exception, the alveoli are of the lacunar type and colloid is absent. The cells are columnar in type, but in places are cuboidal or irregular. Desquamation is seen in many alveoli. Lymphoid infiltration is a marked feature. There is slight fibrosis. The diagnosis was diffuse hyperplastic goitre.

EDITH A., a female, married, aged thirty-six years, was admitted to a surgical ward under the care of Mr. Poate on February 23, 1931,

At that time she had not been well and had had palpitation for two years. She had had shortness of breath for eighteen months. She was "nervy" for one year, but had always been somewhat tremulous and easily upset. She was lacrimose and depressed. She had lost 14·5 kilograms in weight in spite of a good appetite. She had subjective feelings of heat and was perspiring more than normal. She had swollen ankles on walking. Her voice was weak and husky for a year. Her hair was falling out. She had been married one year, no pregnancies had occurred. The menstrual periods were regular, the cycle was thirty-five days, lasting seven days; but she did not lose much.

Previous history: Over a period of from three to four years up to two years before admission she was subject to cramps and had numbness of the fingers.

On physical examination the patient appeared very ill, was very "nervy", fidgety and excitable. Speech was hurried and unsteady. Eyes were staring, definite exophthalmos was present, no von Graefe sign was elicited. Hands were moist and hot, definite tremor was present, hyperextension and trophic changes were also present. The thyreoid was definitely enlarged, both lobes were equally affected; the gland was uniformly soft, very vascular to palpation, and a bruit was audible over the whole of it. There was definite tachycardia. The Wassermann test gave a negative reaction.

On February 27, 1931, the basal metabolic rate was +70%; the basal pulse rate was 104 per minute; the weight was 49 kilograms. She was given three times a day half an ounce of a mixture containing Lugol's iodine (minims 10), bromide of soda (grains 20), tincture of digitalis (minims 20) in each ounce.

On March 13, 1931, the basal metabolic rate was +23%; the basal pulse rate was 80 per minute; the weight was 51 kilograms.

On March 17, 1931, the operation of right hemithyreoidectomy was performed. Pathological report (K.1241,

R.P.A. Hospital) given by Dr. A. H. Tebbutt and Dr. G. Davies is as follows:

The tissue consists of an enlarged right lobe of a thyreoid gland. It measures 7·1 centimetres in a vertical direction, 4·7 centimetres from side to side and 4·9 centimetres in an antero-posterior direction. It weighs 55 grammes. A section in a sagittal direction has been made. This shows a small tumour nodule imbedded in a large amount of thyreoid tissue (*vide* Figure I). The thyreoid tissue has, after formalin fixation, an amber-buff colour and it shows division into lobules, and the interlobular fibrous tissue is increased in the centre of the gland. The tumour nodule is situated so that its most caudal border is 1·9 centimetres from the caudal border of this lobe of the thyreoid, but two grooves in the lower pole penetrate quite deeply into the thyreoid substance, and one of them comes almost into contact with the posterior border of the tumour. A section made from the medial aspect of this lobe of the thyreoid gland shows that another groove comes into close contact with this nodule. The tumour nodule is almost spherical in shape and, where the section passes through it, it measures 0·78 by 0·72 centimetre. It is well defined and is apparently encapsulated. The cut surface is smooth and after fixation cream-coloured.

FIGURE I.

Photograph of the right lobe of the thyreoid gland removed from Edith A., showing the tumour *in situ*. Magnified twice.

Microscopical examination (*vide* Figure II) shows that the tumour is well defined from the surrounding thyreoid tissue. The latter is hyperplastic, the cells being large and cuboidal. Some alveoli are, however, filled with moderately deep-staining colloid and peripheral vacuolation; this is not a feature of the section. The alveoli are mainly large or of moderate size and in many there are infoldings of epithelium. A few smaller alveoli are seen among those of larger size.

The tumour is formed mainly of irregular solid alveoli or double columns of cells which are seen to be irregularly cuboidal in shape. The cell bodies are not well defined. The cytoplasm is eosinophilic, in most cases being granular and in others homogeneous. No cells with clear cytoplasm are seen. There is no definite nuclear heteromorphism or hyperchromatism. Some of the alveoli contain a minute lumen, but no colloid has been formed. The supporting tissue is fibrous and in most places is scanty, but in many places, notably round the larger blood vessels, it is more abundant. There is no attempt at division of the nodule into well defined lobules. In several areas small knots of fibrous tissue have undergone hyaline change. In one place there is a localized area of vascular engorgement and here it is seen that the vessels between the trabeculæ are sinusoidal in character. In other parts only the larger vessels are recognizable as such.

The microscopical structure does not resemble that of any well recognized type of thyreoid adenoma, nor do the cells of which it is built up resemble in detail either the chief or oxyntic cells of the parathyreoid. Benign tumours found in the thyreoid gland and presumably thyreoid in origin are subject to great variations in microscopical structure and there are some that are difficult to classify, such as this one. Is it a very atypical thyreoid or parathyreoid adenoma or has it some other origin? Somewhat similar tumours have been described from this hospital by Tebbutt and Woodhill (THE MEDICAL JOURNAL OF AUSTRALIA, November 5, 1927, Cases XII, XIII and XIV, and Figures XVIII, XIX and XX) and the authors were diffident of exact classification of this particular variety of benign tumour. Most of the parathyreoid "tumours", however, in von Recklinghausen's disease have been indubitably parathyreoid in microscopical structure or recognizable as of this origin. Our conclusion is that the tumour we have described is of doubtful origin, probably thyreoid, possibly parathyreoid.

Subsequent to the operation there were no acute symptoms of tetany, but by April 7, 1931, when the basal metabolic rate was −4·5% and the basal pulse rate 70 per minute, she did not feel well, had difficulty in going to sleep and was very depressed; she had no cramps or numbness; she had a typical fixed expression. Her muscles felt very firm, and her blood calcium was 7·7 milligrammes *per centum*. She was given "Radiostol", ten drops twice a day, with improvement. By August 18, 1931, her metabolism had risen to +64%; the left lobe of the thyreoid was very large, and she had a return of the symptoms of hyperthyreoidism. She had no symptoms of hypoparathyreoidism, but had been taking "Radiostol" intermittently.

On account of the probability of further parathyreoid tissue being present in the remaining lobe of the thyreoid, further operation was considered inadvisable, and in November, 1931, she had a complete course of deep X ray

to the thyreoid gland. (Dosage of ray: 1,600 *r*. (1) to the left side, 1,300 *r*. (1) to the right side; time 82 minutes, 1,640 milliampère minutes.)

On February 26, 1932, the basal metabolic rate was +33%; the basal pulse rate was 100 per minute; the weight was 55 kilograms.

On April 19, 1932, she had all her teeth extracted.

On July 15, 1932, the basal metabolic rate was +61%; the basal pulse rate was 118 per minute; the weight was 56 kilograms. She was very ill, had great difficulty in breathing, and felt she had to breathe deeply. She had typical signs and symptoms of hyperthyreoidism, but she also now had unmistakable signs and symptoms of hypoparathyreoidism. She had carpopedal spasms, severe cramps, numbness and heaviness of the limbs, cravings for different foods, and was extremely depressed and irritable.

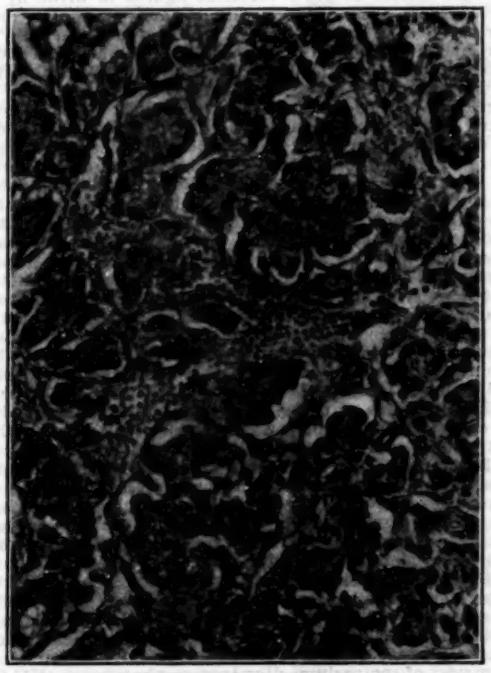

FIGURE II.
Microphotograph of the tumour lying in the thyreoid gland removed from Edith A. (stained with hæmatoxylin and eosin). Magnification: ×250.

On July 22, 1932, she was admitted to the metabolic ward. The experimental diet was commenced.

On July 26, 1932, the first experimental period began.

On July 29, 1932, the basal metabolic rate was +64%; the basal pulse rate was 112 per minute; the weight was 53·5 kilograms. For the first week or two we despaired of keeping her; she was very irritable, difficult to manage, got "worked up" over trifles, and was in a very "nervy" state.

Treatment from July 29, 1932, to August 23, 1932, consisted of rest in bed, no drugs of any kind being used.

On August 23, 1932, blood was taken for calcium and phosphorus estimations. The tourniquet caused a definite Trousseau sign, and cramps of arm. Electrical reactions were tested. "Radiostol" administration, three cubic centimetres daily, was begun.

On August 26, 1932, she felt much better. She had felt very hungry since beginning the "Radiostol"; she felt that the diet suited her. She looked quieter and more natural. The thyreoid gland was still very vascular, but had been getting smaller for the last fortnight. The basal metabolic rate was +31·5%; the basal pulse rate was 100 per minute; the weight was 57·3 kilograms.

On September 20, 1932, the improvement continued. The patient was a very different person. She was much steadier and not at all irritable. She continually told us how well she felt. Her tissues were softer and the ward sister frequently remarked on her looking "natural". She had had no cramps since the first few doses of "Radiostol". She was very anxious to get up. "Radiostol" was increased to six cubic centimetres daily.

On September 28, 1932, "Radiostol" was increased to nine cubic centimetres daily. On the larger doses she felt well, but the sister in charge and several other people who came closely into contact with her did not think she was as well on the larger doses as on three cubic centimetres daily.

On October 6, 1932, "Radiostol" was suspended.

On October 14, 1932, it was noted that for the first few days after leaving off "Radiostol" she felt very depressed and was irritable. This corresponded to a menstrual period which was excessive. (During "Radiostol" therapy she had had a period, but it did not affect her.) After the menses she felt well again.

On October 18, 1932, she left hospital. She was feeling well and did not feel any difference from leaving off "Radiostol". The basal metabolic rate was +34·4%; the basal pulse rate was 100 per minute; the weight was 58·4 kilograms. The thyreoid gland was small and very vascular.

On October 25, 1932, the body was beginning to feel heavy; no cramps were present. She felt well; the tissues were quite firm. She was fidgety, and her expression was somewhat heavy. The thyreoid gland was the same as on October 18, 1932.

On November 1, 1932, the basal metabolic rate was +44·7%; the basal pulse rate was 104 per minute; the weight was 59·3 kilograms. She was "feeling very heavy" though well, but she looked as she did before beginning to take "Radiostol". The most significant fact was that her husband insisted on her beginning "Radiostol" again; he said she was getting hard to live with. Her tissues were very firm. The thyreoid gland was vascular, and she was fidgety. Her electrical reactions were again those of tetany.

Comment.—This was a case of severe chronic hyperthyreoidism associated with definite hypoparathyreoidism.

EMMA B., a female, married, aged fifty-one, has been previously reported (Case VI). Since that report she has attended hospital irregularly, owing to economic reasons. She volunteered to come to hospital for a period of investigation. On admission she was still hyperthyreoidic, and still had signs and symptoms of mild hypoparathyreoidism. On the whole she felt well.

Physical examination revealed a "hangdog" expression. The eyes showed exophthalmos and von Graefe's sign. In the hands there were no signs, but they felt warm. A definite tumour was present in region of the lower half of the left lobe of the thyreoid gland. This tumour was vascular, and a bruit was heard on auscultation. The tissues of the arm were very firm.

On September 9, 1932, on admission to hospital the basal metabolic rate was +28%; the basal pulse rate was 108 per minute; the weight was 52·3 kilograms.

September 12, 1932, was the first day on which specimens were saved.

On September 23, 1932, the basal metabolic rate was +25%; the basal pulse rate was 104 per minute; the weight was 53·3 kilograms.

On September 24, 1932, "Ostelin" therapy was commenced and continued for eight days.

Comment.—This was a case of chronic hyperthyreoidism associated with chronic mild hypoparathyreoidism.

Methods of Analysis.

Results are expressed to two places of decimals for phosphorus and calcium, and to one place for nitrogen. Standard pipettes and flasks were used throughout.

Details of Estimation of Nitrogen.

Nitrogen was estimated by the macro-Kjeldahl method, the ammonia being aspirated by a strong stream of air

into standard one-tenth normal sulphuric acid for an hour with the Kjeldahl flasks standing in boiling water. Neutralisation was affected by aspirating alkali into the acid digest till an indicator turned colour. By allowing some of the indicator to run down the side of the Kjeldahl flask we were always sure the ammonia was evolved. Samples were used that required from 15 to 30 cubic centimetres one-tenth normal sulphuric acid. For food 1·0 gramme samples, for stool 0·5 gramme samples, for urine 5 cubic centimetre samples were found satisfactory. Duplicates were carried out for many months, but as very constant results were always obtained, single samples were then analysed. Titration of excess acid was carried out by one-tenth normal standard alkali.

Preparation of Food and Fæces for Estimation of Calcium and Phosphorus.

1. *Food.*—Five grammes of the dried and sieved food were taken as an adequate sample; this amount was found satisfactory after considerable experimental work. The sample was ashed in a six centimetre diameter platinum dish, over a moderate flame, and took about two hours to be reduced to a white ash. The ash was dissolved in the dish with 15 cubic centimetres of 10% hydrochloric acid (C.P.) and heated till an almost clear solution resulted. Bringing it to the boil for a minute or so was found satisfactory. There was a small insoluble residue which imparted a slight turbidity to the solution. The solution was then pipetted with a five cubic centimetre pipette into an accurately graduated 20 cubic centimetre flask; three and then two cubic centimetres of 10% hydrochloric acid were used for washing and were in turn pipetted into the flask, which was then cooled and made up to volume. Filtration was found to be unnecessary. This solution thus contained 0·5 to 1·0 milligramme of calcium per cubic centimetre and had a normality of 1·0 to 1·5 N. Of this solution five cubic centimetres in duplicate were taken for calcium and 0·5 cubic centimetre for phosphorus estimation. Duplicates for phosphorus were carried out over a considerable time, and then from time to time, but the difference of two analyses was always negligible. Duplicates were always used for calcium, as the possibility of error is much greater than in the estimation of nitrogen or phosphorus. If duplicates were 5% or more out, the tests were repeated. Repetition was seldom necessary.

2. *Fæces.*—Two and a half grammes of the dried and sieved fæces were ashed in a platinum dish as above; 2·5 gramme samples were found to be adequate to obtain representative samples. Fæces take about one hour to ash to whiteness; both the food and fæces when heated give off inflammable gases which readily ignite; there is but little odour. The ash is dissolved as for food, from 40 to 50 cubic centimetres of 10% hydrochloric acid being used and made up to 50 cubic centimetres; the final solution contained from 0·5 to 1·0 milligramme of calcium per cubic centimetre and had a normality of 1·0 to 1·5 N. Of this solution five cubic centimetres in duplicate were taken for calcium, and 0·5 cubic centimetre for phosphorus estimation.

Preparation of Urine for Estimation of Calcium.

Direct methods of precipitating calcium from urine[Sato] [Clem] did not prove satisfactory in our hands. The urine was always evaporated to dryness and organic matter destroyed. Organic matter was destroyed as follows: From 75 to 200 cubic centimetres of urine *plus* 15 to 20 cubic centimetres of calcium-free nitric acid (a locally made commercial nitric acid was found to be satisfactory) were evaporated to dryness in a Kjeldahl flask of 300 cubic centimetres capacity; when dry, hydrochloric acid and nitric acid were added till a perfectly white ash that was almost completely soluble in 20 cubic centimetres of dilute hydrochloric acid (10% approximately) was obtained. The solution was brought to the boil and pipetted into a 30 cubic centimetre flask. The sides and bulb of the Kjeldahl flask were washed with five and four cubic centimetres of dilute hydrochloric acid and added to the measuring flask. The latter was cooled and made up to volume. The final solution contained 0·5 to 1·0 milligramme of calcium per cubic centimetre and had normality of 1·0 to 1·5 N. Of this solution five cubic centimetres were taken in duplicate for analysis.

Preparation of Urine for Estimation of Phosphorus.

The urine was always kept acid pending analysis. Of the carefully mixed urine of the period, 0·5 or 1·0 cubic centimetre amounts were found to be satisfactory. Duplicates carried out over a long period always agreed to within 1%.

Details of Method for the Estimation of Phosphorus in Food, Fæces and Urine.

A good deal of preliminary work had to be done to get a simple and satisfactory method, by which many analyses might be performed at a time. The stannous chloride method of Youngbury[Youn] did not give us satisfaction, and we were unable to get theoretical figures in urine by the use of Fiske's method.[Fiske] The method which we found extremely satisfactory is practically that described by Martland and Robison[Mart] for total and soluble phosphorus in blood. This is a modification of Brigg's[Brigg] modification of the Bell and Doisy method.[Bell] In the case of the solutions obtained from food and fæcal ash, 0·3 cubic centimetre of concentrated sulphuric acid (C.P.) was added to 0·5 cubic centimetre of the solution in a 30 cubic centimetre "Pyrex" tube. These test tubes were drawn out at the junction of the upper and middle thirds and graduated for 20 cubic centimetres. A constricted tube of this nature allows of somewhat stronger heating than a straight tube without loss of phosphoric acid, and the subsequent operations may be carried out without further transfer of the material for analysis. The test tubes were then heated over a small flame till sulphuric acid began to condense on the sides of the tube below the constriction. By this means excess hydrochloric acid, which seems to interfere with the development of the characteristic blue colour, is removed. The test tubes are then allowed to cool slowly. Urine was treated as follows: To 0·5 or 1·0 cubic centimetre of urine in a "Pyrex" test tube as above 0·3 cubic centimetre of concentrated sulphuric acid (C.P.) was added, and the test tube heated till charring was well advanced. Perhydrol[1] was then added drop by drop from a capillary pipette with further slight heating, till a colourless solution resulted. After cooling, the sides of the test tube were washed down with a few drops of distilled water and the last trace of hydrogen peroxide was removed by again heating the test tube till sulphuric acid began to condense on the sides of the tube. Further steps were then as described by Robison. The standard in all cases was three cubic centimetres of a solution containing 0·00001 gramme of phosphorus per cubic centimetre, prepared from a stock solution containing 4·39 grammes of potassium dihydrogen phosphate (KH_2PO_4) per litre, preserved with chloroform and kept in a refrigerator. There is no advantage in having multiple standards, as three cubic centimetres made up to 20 cubic centimetres and set at 20 millimetres give satisfaction with any depth of colour in the unknown from nine millimetres to 30 millimetres. When the unknown is stronger than a reading of nine millimetres the test was repeated, less of the solution being used for analysis. The standard was estimated for phosphorus by the classical Neumann method[Neum] and found to contain 4·394 grammes of potassium dihydrogen phosphate per litre.

A comparison of phosphorus content of the same solutions was made by the Neumann method and colorimeter method, with the following results:

	Neumann Method.	Colorimeter Method.	Difference.
Urine	2·670 grammes	2·630 grammes	−1·5%
Stool	0·833 gramme	0·825 gramme	−1·0%
Food	3·630 grammes	3·550 grammes	−2·2%

[1] The perhydrol was specially prepared by the kindness of the Crystal Chemical Company of Sydney, and was phosphorus-free. This had no stabilizer, but retained its hydrogen peroxide indefinitely if kept at 8° C.

Estimation of Calcium in Food, Fæces and Urine.

Choice of Method.—Macro-analytic methods are very accurate, but a large sample has to be ashed for analysis. The micro-method of titration of oxalate with N/100 permanganate has been criticized by van Slyke and Sendroy,[136] who proposed a method based on estimating the amount of carbon dioxide liberated from calcium oxalate. Fiske[47] advised precipitation as oxalate, ignition to oxide and titration with N/100 acid, and claims great accuracy. These methods proved too time-consuming for our purposes, and a modification of the Kramer and Tisdall method for serum analysis[77] was developed.

Method Adopted.[1]—The calcium present in the solutions prepared as described above is precipitated as oxalate in a centrifuge tube, and titrated with N/20 potassium permanganate solution from a five cubic centimetre burette, graduated in one-hundredths of a cubic centimetre.

Reagents required are as follows: (i) Standard potassium permanganate (approximately N/20). The solution is boiled and then standardized against purest oxalic acid $(COOH)_2.2H_2O$. If stored in a dark bottle this solution maintains its normality indefinitely. (ii) Oxalic acid solution (C.P.), 10% approximately. (iii) Ammonium chloride (C.P.). (iv) Ammonium hydroxide solution, approximately 4N. (v) Methyl red indicator (saturated alcoholic solution). (vi) 2% ammonia solution (wash fluid). (vii) 10% sulphuric acid (C.P.).

Technique.—Five cubic centimetres of the calcium solution are pipetted into a centrifuge tube of about 15 cubic centimetres capacity, diameter about 1·5 centimetres, tapering from the middle to the closed end. (Great care is necessary in washing these tubes to insure freedom from grease. We brushed with hot water and sand soap, then rinsed very thoroughly and left the tubes full of acid bichromate solution overnight.) Approximately 0·3 gramme of ammonium chloride is added and dissolved by stirring, and then 1·5 cubic centimetres of 10% oxalic acid solution and one drop of indicator. On adding 4N ammonia solution with constant stirring until a pH of 5 is reached, the calcium oxalate precipitates quantitatively at room temperature. The final solution should be pink. The stirring rod is washed with a fine stream of distilled water, and the tube is allowed to stand for one or two hours and then centrifuged for ten minutes at 2,500 revolutions per minute. The supernatant liquid is poured off, the tube inverted and drained for twenty minutes on to a Terry towel, and then dried with absorbent paper to a distance of about five centimetres from the mouth. Five cubic centimetres of 2% ammonia are run in very carefully, with the tube held almost horizontally and rotated to wash the sides thoroughly; the precipitate is not disturbed. After standing for half an hour the tube is centrifuged for ten minutes, inverted, drained for twenty minutes and wiped as before.

The calcium oxalate is now ready for titration. It is dissolved in five cubic centimetres of 10% sulphuric acid, heated by standing the tube in boiling water, and titrated with N/20 permanganate. During titration the solution is agitated with a stirring rod.

Checks on the Method.—Test solutions of calcium oxalate were modified by the addition of varying quantities of magnesium phosphate and sulphate ions. Magnesium and phosphate in considerable concentration did not affect the results, nor did sulphate, provided the calcium sulphate did not approach the limit of saturation. The concentration of ammonia used for precipitation, and the presence or absence of carbonate in this ammonia, were not found to affect the accuracy. Leaving the tubes to stand one hour was found ample to cause the whole of the calcium to precipitate. Variations in curvature of the end of tubes, drainage for longer than thirty minutes, longer contact with the wash fluid, or washing twice, were not found to affect the result appreciably.

Accuracy.—Standard solutions prepared from pure naturally-occurring crystalline calcium carbonate (calcite, $CaCO_3$) gave theoretical results.

[1] We wish to acknowledge the assistance of Dr. A. Bolliger in developing this method.

Morris, Nelson and Palmer[120] state that the ash from organic matter insoluble in hydrochloric acid still contains appreciable amounts of calcium, and by fusing this ash with anhydrous sodium carbonate they obtained yields as much as 20% higher. We fused the insoluble ash from twenty grammes of dried food and from twenty grammes of dried fæces with anhydrous sodium carbonate, then dissolved the fused mass in hydrochloric acid. This was tested for the presence of calcium, but contained negligible amounts. Similarly, when the insoluble ash was treated with *aqua regia* no residual calcium was found.

The method gives results of satisfactory accuracy, it is comparatively simple and expeditious, and allows a large number of analyses to be performed at one time.

Blood Chemistry.

Blood was obtained before breakfast by venipuncture, and run directly into paraffin-coated centrifuge tubes. The tubes were prepared as recommended by Keynes.[70] The tubes were kept on ice and centrifuged as soon as possible. If a plasma clot formed, which was the rule, the clot was pushed down with a broad glass rod and the tube was again centrifuged. Our object was to obtain a cell-free serum as rapidly as possible, but the method may have some effect on the serum calcium; Stewart and Percival[114] discuss this problem. We carried out estimations of calcium and phosphorus on a series of pathological conditions to act as controls. On the whole the method is not satisfactory, owing to the rapidity with which a plasma clot forms, and the poor yield of serum or true plasma that is obtained.

Calcium was estimated by Clark and Collip's[28] method, except that twenty-four hours were allowed for the calcium to precipitate. Inorganic phosphorus was estimated by Robison's[84] modification of Brigg's method, except that the coagulated material was filtered through a small Gooch filter using number 40 Whatman filter paper. Slight suction was found to cause the most rapid filtration. We used a Gooch filter because filter paper has the power of absorbing an appreciable amount of phosphate.

Estimation of Basal Metabolism.

Expired air was collected in a Douglas bag and analysed in a laboratory pattern Haldane.[80][43] The test was performed twice a week during the experimental period, and always fifteen hours after food. The patients had to be wheeled only fifty yards, so that there was very little disturbance. Du Bois standards[42] were used, except for the case M.R., a child of twelve. In this patient the results are given as calories per square metre per hour, because we consider it impossible to compare one child with another at this early age. (See discussion by Talbot,[130] Du Bois,[42] Gierring,[21] Dinsmore.[41]) We had hoped to make her her own control, but her hyperthyreoidism proved intractable to X rays, and at the time of going to press she could not be got into a satisfactory state for operation.

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REST AND MOVEMENT.[1]

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THIS address is entitled "Rest and Movement", and it is proposed to discuss the value and the indications for use of these therapeutic agents in the treatment of joint lesions.

Joint lesions vary considerably in severity and in importance both in present and future. At the bottom of the scale is the slight sprain; there follows dislocation, then come adhesion and stiffness of joints, chronic aseptic inflammation, then all grades of bacillary inflammation, from those of a mild or chronic nature to the peak of acute suppurative arthritis.

In the simple aseptic traumatic lesions the objective of treatment should be the restoration to the joint of its normal functions of movement and weight-bearing. But in the inflammations of bacillary origin, since their course is fraught with grave danger of local abscess and systemic infection, the aim should be the safety of limb and life with articular function a very secondary consideration.

It is necessary, then, to determine at the outset, from a knowledge of the potentialities of a particular joint lesion, whether it is obligatory for us to consider its treatment from the standpoint of danger to limb and life, or whether we may concentrate on the restoration of the joint. The therapeutic agents under discussion, rest and movement, admit also of gradation. The theory of relativity is applicable to them.

Rest and movement are at opposite ends of a scale. Rest may be regarded as the lowest form of movement; movement as the worst form of rest.

[1] Read at a meeting of the Victorian Branch of the British Medical Association on July 22, 1933.

My purpose is to show that as the lesions of joints vary in degree, so the therapeutic agents may be graded to fit them. Very frequently our knowledge and experience enable us to determine what degree of movement can be allowed to a particular lesion, but too often, owing to inability to understand fully its pathological nature, we must resort to the test of trial and error and modify the treatment accordingly.

The subject will be discussed under the four headings of rest, modified rest, passive movement and active movement.

Rest.

Rest, as we use it in treatment, is a comparative term implying a degree of depression of the anatomical and physiological function of the joint.

In the superlative degree of rest the function of the joint is completely obliterated by bony ankylosis—no movement of any kind can occur.

This is the objective in all forms of destructive arthritis. It may be artificially produced by the operation of excision and arthrodesis or naturally acquired as the result of the destructive lesion.

Destructive arthritis comprises lesions in which, though the course be rapid and acute, as in suppurative arthritis, or slow and chronic, as in tuberculous and allied forms or in the degeneration of osteoarthritis, whether senile in nature or initiated by trauma at a younger age, all ultimately and inevitably end in erosion of cartilage and the contact of bone with bone.

For these lesions superlative rest means an end to their dangers and relief from pain. When bony ankylosis is attained, then, and not until then, are the conditions safe and painless.

Complete Rest.

The next grade of rest, which we may term "complete rest", is that in which, though the joint still exists, its functions are depressed to their lowest point.

The requirements for this method of treatment are as follow:

1. It is essential that the joint be nursed in a position of comfort. This position will be short of the extreme limits of movement, for it is at these extremes that some articular structure ligament or tendon is tense and strained. A useful guide to the posture of comfort is the atttiude of a joint during sleep.

2. The function of weight-bearing must cease; hence the patient must be in bed.

3. The effect of gravity must be nullified by balanced suspension of all segments of the limb.

4. All movements must be prevented, so the joint must be adequately splinted to stop flexion and extension, rotation, abduction and adduction.

5. All muscles acting on the joint must be set at rest by control of the muscle from beyond its origin to below its insertion. This necessitates fixation of the joint above and below the affected articulation.

Destructive Arthritis: Acute suppurative; Tuberculous; Osteoarthritis: Senile, Traumatic	Superlative (Ankylosis)	Rest
Acute Arthritis; Perforating Wounds (prophylactic); Subacute Arthritis	Complete	Rest
Chronic Bacillary Arthritis; Chronic Aseptic Traumatic Arthritis	Restriction of range; Hoppling of joints	Modified Rest
Stiffness; Adhesions of joints	Passive movement	
Dislocations; Sprains	Active movement	

6. To prevent the harmful effects of rubbing or hammering of one joint surface on the other as the result of muscle spasm, extension must be applied to one segment of the limb so as to pull the articular surface apart.

Such a condition of rest is called for in the treatment of perforating wounds of joints and of the various forms of acute pyogenic arthritis.

A perforating wound of a joint bears much the same relation to a simple joint lesion as the compound fracture does to one of the simple variety.

The simple bone or joint lesion carries with it practically no risk, whereas when the trauma is compound in bone or joint the whole picture is altered to one of the gravest prognosis.

The prevention of sepsis in the compound fracture or of perforated wound of a joint is of the utmost importance, and treatment must be directed towards it immediately.

Of the various measures that are of benefit in the prevention of septic arthritis, none, in my opinion, is more effective than complete rest of the type described above.

Active Movement.

Active movement implies voluntary contraction of muscles, with or without visible movement of the joint structures.

It is a beneficial agent in that: (i) It keeps muscle in tone and prevents the wasting of disuse and so helps to maintain the stability of the joint. (ii) It relieves the stasis of venous blood and lymph fluids and evokes a better arterial circulation. (iii) It aids in the rapid absorption of extravasated blood and traumatic exudates and so hastens repair. (iv) It prevents the formation of adhesions.

Nature's danger signal is pain, and within the limits of pain active movement never does any harm and is the method of choice in the treatment of aseptic traumatic lesions of joints, such as recent sprains and dislocations.

The other safeguard in its use is the rule that if the range of movement of a joint increases daily, then the treatment is correct, but if the range decreases it is a warning that rest is indicated.

Passive Movement.

Passive movement is movement imparted to a joint by the operator. It is movement directed against the resistance of adhesion or stiffening.

Passive movement in the treatment of an adhesion is sudden and forceful, and is designed to rupture the restraining band that results from a sprain, and so the after-treatment is that of a sprain and consists in active movements directed towards securing full range of movement.

That is manipulative surgery, and for an account of it Bankart's "Manipulative Surgery" should be consulted.

Passive movement may, on the other hand, be applied repeatedly almost every day in a much less forceful manner designed to stretch gradually, rather than rupture suddenly, shortened structures. It is used where the stiffening is more extensive, as in the late results of rheumatoid arthritis and of periarticular sepsis. This type of passive movement may be usefully referred to as treatment by mobilization and massage. Pern[1] has written a useful account of his experience with this method.

The healing of an abdominal operation wound with all the advantages of fixation and coaptation of tissues by many sutures is not in general complete within two weeks. It is therefore not to be expected that tendinous or ligamentous healing devoid of these aids will take place within this time.

Passive movement is contraindicated until sufficient time has elapsed to allow of complete healing of a traumatic lesion or subsidence of an inflammatory one. Disregard in this respect must lead to further trauma of a healing lesion or flaring of a smouldering inflammatory lesion.

At all times the use of passive movement must be controlled by observing the rule that if the range of active movement decreases, then rest, not movement, is indicated.

Modified Rest.

At the extreme limits of movements of a joint some of the articular structures are in a state of tension or strain, that is, a condition of unrest. If, therefore, we prevent the joint from moving to its extreme limits, we have restricted its range, eliminated some strain upon its structures, and thus imposed on it a certain degree of rest.

We may term this method of treatment modified rest or restriction of range or, better still, hoppling or fettering the joint. Hoppling the joint is indicated in the chronic inflammation of ligament or tendon.

Suppositious Cases.

And now let us examine a few patients who present themselves and require either rest or movement.

Case I.

A young man has sprained his ankle this afternoon at football. About the external malleolus there are swelling and discoloration and tenderness, and on movement, pain, indicating that he has torn the anterior and middle bundles of his external lateral ligament.

First it is necessary to prevent over-stretching of the torn ligament, and for that purpose we apply an inch wide strip of adhesive strapping, so placed that it prevents the foot from going to the inverted and plantar position.

Next it is necessary to get rid of the effusion of blood and traumatic exudate as quickly as possible. This requires some form of elastic pressure, and we may bandage over the area a rubber sponge or a very thick layer of brown cotton wool, which retains its elasticity much longer than and does not pack like white wool.

Next we encourage the patient to put his ankle through various movements up to the point of pain.

And finally, because his articular cartilages have not been injured, that is to say that the weight-bearing surfaces have not been traumatized, we urge him to walk on his recently damaged ankle.

That is the treatment of a recent sprain by active movement.

Case II.

The next patient is a youth who "did his ankle in four months ago and the quack put it in plaster for a month". Since that time there has been a feeling of insecurity in his ankle joint, and on certain movements there is pain. On testing the ankle movements we find that in some directions the range is quite normal, but in others pain is produced and the range is limited.

He has adhesions of the torn ligaments and his treatment consists in giving him an anæsthetic and manipulating his ankle, breaking the adhesions. Then he will be in the same state as the first patient, suffering from a recent sprain, and his treatment will follow along those lines—active movement to prevent the reformation of adhesions.

Case III.

The next is a lady of fifty who, ten years ago, sustained a fracture involving the ankle joint. For ten years she has suffered pain, which has been increasing, and a loss of range of movement, which has also been increasing; there is pain and stiffness of a progressive nature.

As the X ray films show considerable narrowing of the joint space, it is obvious that the articular cartilages are gradually being eroded, that she is suffering from degenerative arthritis—an osteo-arthritis initiated by trauma.

She is a healthy woman and has twenty years to live. There is, by natural means, no end to her pain. Let us then apply the principle of superlative rest of this ankle joint; let us ankylose it by operative means and for all time put an end to her pain and suffering.

Case IV.

The next patient cannot play golf—at least he can play four holes with gradually increasing pain in his big toe joint. At about the sixth hole pain and disability become so great that he has to desist. He has an area of traumatic inflammation on the dorsum of the metatarso-phalangeal joint, and as it goes into the hyperextended position the movement gives rise to pain. Let us hobble this joint; let us prevent it from going into hyperextension by giving him a rigid soled boot; put him into a pair of heavy military boots studded with sprigs, with a sole that will not allow of bending, or split the sole of his golf shoe and insert a thin steel plate underneath his big toe joint. That will give modified rest to his big toe joint, limit the range of movement and put an end to his pain.

Case V.

Nine months ago the next patient sustained an injury to the calf of his leg, tore fibres at the junction of the muscles and *tendo Achillis*—a tennis leg. At times he has pain and tenderness and some swelling about this area, and as the day goes on it gets worse. He says they told him to use it; that is the treatment by active movement; but what they forgot to tell him was that he was to use it only within the limit of pain, and he has been over-stretching it. In this traumatized area there is a tender thickening at the junction of muscle and tendons; it is the site of a chronic aseptic traumatic inflammation resulting from repeated injury and tearing of healing tissues. In the act of walking he puts the sound leg ahead of the injured one and so forms an acute angle between foot and leg on the injured side and over-stretches his calf muscles. Let us teach him to walk up to the injured leg, but not beyond it, so that the angle between foot and leg is never less than a right angle; or, better still, let us raise the heel of the boot on his injured side by 3·75 centimetres (one and a half inches), so that now in standing erect foot and leg form an obtuse angle. He may now walk in the natural manner without stretching calf muscles, and slowly his tenderness and pain will disappear.

Case VI.

A youth has had a pain in his knee for four months; it is better in the morning when he rises; the knee aches at the end of the day; the pain is deep in his knee joint. He has a chronic traumatic inflammation of his joint.

Inquiry into the manner of its production shows that whilst playing football his foot slipped ahead of him and a player fell across the front of his knee, thus hyperextending it and doing damage to the crucial ligaments which limit hyperextension.

On examining it we notice that whilst it appears to be fully extended it just falls short of that

position in comparison with the normal joint; when we attempt to push it back into the fully extended position we notice the elastic recoil that is due to Nature's protective spasm of muscles. Should we put a back splint on that patient? No, for that will be merely straining and inflaming the damaged structures. Let us put a Marsh's cage splint on his knee with a stop at the hinge which will check extension at 20° short of the limit of that movement, that is, hoppling his joint and applying the principle of modified rest.

Case VII.

The next youth complains of similar symptoms, especially on going up and down stairs. His pain is produced in the knee cap or at the lower end of it. Inquiry into the method of production of injury shows that whilst playing football the injured foot skidded ahead of him so that he was falling whilst his body and his knees were in a bent position. Endeavouring to prevent the fall, he made a violent contraction of his quadriceps in an attempt to bring his body into the vertical and stable position and at the moment suffered a pain in his knee. That is the type of injury that frequently results in a fracture of the patella by muscular violence. In this case the bony patella has withstood the strain; nevertheless violence has fallen on some other part and he has suffered a trauma either of his patellar articular cartilage or of the tendinous fibres inserted into his patella. Hence any attempt to walk up and down stairs brings under strain the area which has been damaged by the original violence; and so we must prevent the patient from carrying his body weight on a flexed knee joint. We must fit a back splint to his knee and we must teach him to walk up and down stairs by carrying his damaged limb behind him on going up and by putting it on the lower step first on coming down.

So we shall relieve the traumatized area from further damage and allow healing to take place.

Case VIII.

The next patient has suffered dislocation of the shoulder, which has been successfully reduced, and we wish to institute treatment by active movement. The tissues which have been damaged are the inferior portion of the capsule of the joint, and we must encourage him to put his arm into abduction. He is afraid to do this in the erect position, because it means lifting the whole arm against gravity, so we must trick him into putting his arm into the abducted position. We do this by letting his arm hang by his side and then slowly tilting his body laterally whilst letting the arm swing, and we find that we have opened the angle between the arm and thorax and have secured the movement of abduction; or we may cause him to lie on a smooth table, and with the effect of gravity thus removed induce him to move his elbow slowly into the abducted position.

Case IX.

The next patient is a sailor whose arm was jerked by a rope into a position of abduction. This injury occurred some months ago and should in the ordinary course of events have produced a dislocation of the shoulder. It just failed to do so, but it traumatized the tissues of the under-surface of his joint. He did not consult a doctor; he carried the arm in a sling; now he complains of disability of his shoulder joint. He cannot actively abduct his shoulder to a right angle, nor can we passively abduct it, although it swings backwards and forwards to its full extent; we find also there is a loss of the rotary movement.

He has adhesions about his shoulder joint and will be treated by anæsthetization and manipulation of his shoulder joint in order to break the adhesions, and then he will be encouraged to put his arm daily through the full range of movement.

Case X.

Number ten is a patient who has a pain in the shoulder also. The pain is annoying rather than a severe pain and almost disappears for a time and then becomes as bad as ever. He can raise his arm from his side through the abducted position to the position of full extension, but there is a painful arc of movement from about 40° to 90° and the pain is felt in both the upward movement and the downward movement through the same arc.

X ray examination reveals a calcareous shadow about the greater tuberosity at the humerus. This patient has a tendonitis of the supraspinatus tendon. It probably is the result of a minute tearing of fibres with the formation of hæmatoma and calcification as the result of chronic traumatic inflammation. He has to be educated to dodge the painful movements; he has to be taught that he cannot abduct his arm and put his arm in his sleeve; he must put his coat on to his arm; he must be trained to avoid the movements which give him pain and so to rest the injured tendon. In addition we may put a strip of plaster from front to back across the middle of the belly of his deltoid muscle; now, when he attempts to contract his deltoid, he is made aware of the tightness and is reminded that he must not carry out this movement.

Case XI.

The next patient has had for four months a swollen, stiff and painful finger. The finger was stubbed on the end while he was playing football; it was forced into the hyperextended position, and since then there have been gradually increasing stiffening, swelling and thickening. This joint must be splinted in order to give it rest, but it certainly should not be splinted in the fully extended position. Rest for this joint demands a position of comfort, and that position is one of semiflexion. Too often do we see that injured finger bound to a straight piece of wood, a position with tension on the damaged structure.

Case XII.

The next patient is a typist who knows that she has neuritis of the arm because she has been told so by three doctors, who have all given her aspirin

without much benefit. She gets an ache and pain up and down her arm, but a closer inquiry reveals that the maximum pain is about the elbow joint, and in particular about the external epicondyle. Further examination shows that it is movement such as lifting the teapot with the arm extended, turning a door handle, or even grasping it, which gives her pain which is situated somewhere about the external epicondyle. She is told that she has what is known as "tennis elbow", but she says she has never played tennis in her life.

The term "tennis elbow" is misleading, for the condition may occur in patients following all kinds of occupations—in blacksmiths, plasterers, clerks *et cetera*. It really is a tendonitis of the extensor muscles close to their origin from the external epicondyle. Resting the elbow does not cure it, for the patient can still extend the wrist and metacarpal of thumb, and the thumb itself, and it is these movements which keep the condition going. It is better to disregard the elbow end of the extensors and to hopple them at the other end by the wearing of a glove in which the thumb and index compartments are sewn together. Prevented now from abducting the thumb, which is the first and the essential movement in all grasping actions, the patient does not attempt those minor movements which, repeated almost unconsciously throughout the day's work, have maintained the traumatic inflammation of the tendons about the elbow and rendered it chronic. Given modified rest in this way, the condition starts on the road to recovery.

Reference.

(1) H. Pern: "The Treatment of the Joint Lesions of Arthritis Deformans", THE MEDICAL JOURNAL OF AUSTRALIA, May 13, 1933, page 573.

SITE PREDISPOSITION TO CANCER.

By R. DOUGLAS WRIGHT, M.S. (Melbourne),
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STATISTICS showing a varied incidence of carcinoma at different anatomical divisions of an organ loosely imply that the cells of the several parts assume a neoplastic habit with varied ease. Each cell of the organ may be the locus of neoplasm formation and is thus at risk. Do some more readily become neoplastic than do others, or is there a fallacy in argument from site incidence?

I have remarked that all the cells are at risk. In any one organ there will be an approximately constant number of these cells per unit area. The question of predisposition of the cells to onset of malignant disease will therefore be more easily answered if we consider the area of the anatomical part involved relative to the total area of the epithelium at risk and relative to the incidence of carcinoma in each particular portion.

The large bowel is an organ excellently suited for this analysis. External irritation does not affect certain parts, as in the case of the tongue, and it is sufficiently large for accurate anatomical location of the origin of malignant disease; the stomach is too small for this observation to be made accurately; eccentric spread in a small organ obscures the actual locus of origin.

The next step in the analysis is simple. From standard text books the diameter and length at moderate distension of each part of the colon are easily ascertained; the surface area (excluding foldings, which are practically regular throughout the bowel and so need not trouble us) is in proportion to the product of these two. From these figures we find the percentage of the total mucosal area of the bowel in each anatomical division. As the flexures are not accurately assessed as a rule, they are included in the transverse colon. When in the table we compare the percentage of area with the percentage of local incidence of carcinoma (from the statistics of Körte, Petermann and Anschutz, quoted by Ewing,(1) and Kausner and Clarke(2)), we find a remarkable correspondence between these, except in the case of the caecum, in which the incidence of carcinoma is two and a half times that estimated by probability relative to surface area.

Part.	Percentage Area.	Percentage Carcinoma.
Caecum	6	15
Ascending colon	13	12
Transverse and flexures	35	30
Descending colon	8	5
Sigmoid colon	38	38

Thus it is not the cell of the sigmoid colon, in which is the greatest incidence of carcinoma, that is "predisposed" to cancer, but the cell of the caecum.

References.

(1) Körte, Petermann and Anschutz, quoted by Ewing: "Neoplastic Diseases", Third Edition.
(2) Kausner and Clarke: "Analysis of 104 Cases of Carcinoma of the Large Intestine", *American Journal of Cancer*, Volume XVI, 1932, page 940.

Reviews.

CANCER AND CIVILIZATION.

PROFESSOR J. B. S. HALDANE devotes one of his delightful essays to "Science for Amateurs". His suggestions include, *inter alia*, meteorology, mathematics, natural history, but he does not mention cancer research. Dr. John Cope has, however, endeavoured to fill this space to the extent of nearly three hundred pages.[1]

His plea is for a study of cancer and its causes by the methods of the eighteenth and nineteenth centuries, not by the laboratory and statistical methods of the twentieth. Warbury's discovery of the glycolytic powers of malignant cells means nothing to him, nor the fact recently brought out by American statistics that the greatest factor discoverable in the production of gastric carcinoma is hurried food-taking. Irritation he dismisses in a few lines, and experiments on mice and fowls as having no bearing on the subject. Instead, he turns for causation to tissue degeneration, and here at times he is on firmer ground. The breasts which give no suck may be, it is true, more prone to cancer than those of the mother who has nursed

[1] "Cancer: Civilization: Degeneration", by J. Cope. London: H. K. Lewis and Company, Limited. Super Royal 8vo., pp. 310, with 55 illustrations. Price: 15s. net.

five or six children, and the virgin uterus (apart from post-traumatic cancer of the cervix) more than the parous. But unfortunately he strays far from his subject. A long discussion on cancer of the digestive tract ends with the conclusion that its cause is "civilised" or sophisticated food. The same causes by a "house that Jack built" train of reasoning are blamed for gastric ulcers, which lead again to precancerous degeneration and ultimately to cancer.

> The quality of resistance should be raised. To do this the whole mechanism of digestion from the mouth to the anus must be given more strength and endurance . . . . Exercise, and exercise alone, can enrich the parts with blood, and bring to them those mechanical impulses and that access of growth which shall enable them to become efficient.

Cancer of the reproductive (Dr. Cope prefers "racial") organs is equally imputed to diminished child-bearing, employment of women, athletics, the emergence of the neuter and contraception. But he takes two long and rather inflated chapters to discuss this.

He then spends some hundred or so pages of discussion on cancer prevention. Apparently the measures to be adopted are: (i) Woman to be sent back to her proper place—the home—to bear, suckle and train children. (ii) Early marriage and child-bearing. (iii) Anything that makes for hardihood, endurance and other youthful qualities. (iv) Improvement of social conditions. (v) In short, "to exercise those organs which in the circumstances of our civilization are now being insufficiently used and to cause their invigoration and rejuvenation". (But Voronoff is nowhere mentioned.)

Finally, there are fifty pages of completely irrelevant discussion on causes of national degeneration; in these the author airs his medical, political and economic opinions to his heart's content.

Among all the welter of quotations (there is not one complete "reference" in the book), opinions, assumptions, and half truths there is a certain amount of real thought and constructive suggestion; but it takes much search to find it. The searcher will indeed encounter much that is interesting and provocative, but it is hardly fair to relate it all to cancer.

Whether good or bad, so-called civilization (apart from an international cataclysm) is to be lived with; what we must devise is means of doing so.

The city baby may no longer get in its slum the sunlight, vitamins and fresh air of the country baby; but we can give it all these by a little forethought and design; they are not essential features of city life. Dr. Cope, on the other hand, would have us tear down our cities and return to Nature, to raw meat, raw fruit, and large, early families.

The printing and paper are delightful; the diagrams are mostly applicable, and the index is good. There is, as has been noticed, practically no instance of a completely identifiable quotation nor any list of references.

PHYSICS.

THE sub-title of the text book on physics by J. S. Rogers implies that the students who make use of it have already undergone a course of instruction in the fundamental principles of physics and are now to use this book to assist them in gaining a more complete and detailed knowledge of the special sections of this science which find application in the art of the modern medical practitioner.[1]

It is on this understanding only that we are prepared to give an unqualified blessing to a course of instruction in physics, even in a professional faculty of a university, in which the logical completeness and unity of the subject are sacrificed to considerations of a too narrow utilitarianism.

> "Now listen to me", said a prosperous gold-miner when entering his son for a course of instruction in mining at a well known Australian school of mines, "I don't want the lad to waste his time learning geometry and drawing and chemistry and all such useless stuff. I want him to get right on to gold". This extreme practicality of outlook in matters educational is by no means confined to gold-miners. Similar views are sometimes expressed by members of more learned professions. There always has been and always will be conflict of ideals between the academic and the professional viewpoints in education, the former aiming at giving a thorough scientific foundation to the technical knowledge of the student, the latter at teaching him only what is practically useful. The book before us is written from the practical viewpoint, but within the limitations that such a viewpoint imposes the treatment is thoroughly scientific; and it must be admitted, even from the academic aspect, that there is much to be said for a selection of material which engages the interest of the student by reason of its relevance to his future professional activities.

For the book itself we have little to say that is not commendation. Both in the choice of subject matter and in the clearness and accuracy of exposition it is distinctly superior to some books of a similar kind which have in recent years been published on the other side of the world. It seems doubtful, however, whether the two first chapters, in which a rapid historical *résumé* of the whole history of physics, necessarily in an extremely condensed form, is attempted, and which are incongruous with the general scheme of the book, are worth while. Presumably the student has already had the opportunity of gaining acquaintance with the general historical development of physical science in his preliminary course of study, and, if so, this repetition is superfluous. In any case a mere chronicling of events is a crude and inartistic way of teaching history of any kind, which, fortunately for the youth of today, is at last falling into discredit even with professional historians. In place of these two chapters we would prefer to see some space devoted to such fundamentals of scientific method pertinent to medical science, as, first, the law of dimensions, the profound bearing of which on questions of organic structure and function is convincingly demonstrated in Professor D'Arcy Wentworth's fascinating book, "Growth and Form"; secondly, the elements of statistical method as applied to physical measurements in the field of human anatomy (for example); and thirdly, perhaps, some illustrations of sense illusions such as are given in R. W. Pohl's recent book on mechanics and acoustics. These last have a special interest for the student of human medicine, for whom, moreover, it is doubly important to realize that "the sensation of colour (along with other sensations and feelings) is not an object of the science of physics, but rather of psychology or of physiology".

These remarks are offered merely by way of suggestions for a future edition. Within the scope which the author has set for himself there seem to be few omissions of any note. Is it safe, however, to assume that the average medical student understands the ordinary mercury thermometer, or its clinical modification, so completely that no reference to it is necessary? We do not think so. The clinical thermometer is still by far the most important instrument used in diagnosis, and, simple though it be, this importance should be emphasized by its inclusion in the book. Thermo-electric and resistance types of thermometer are also finding increasing use in medical diagnosis and research, and for this reason deserve at least brief mention.

Another very important instrument, the stethoscope, though mentioned, is not discussed, and the electrocardiograph is not referred to. These slight omissions, which, after all, may have been made after consideration and for very good reasons, do not appreciably detract from the value of the book as a whole, a perusal of which fully justifies the publishers' hope that it "will have a definite appeal to medical practitioners as well as to students".

The book is excellently printed, and the very small number of typographical errors bears witness to careful proof-reading and type-setting.

[1] "Physics for Medical Students: A Supplementary Text Book", by J. S. Rogers, B.A., M.Sc., F.Inst.P.; edited by T. H. Laby, F.R.S.; 1933. Melbourne: Melbourne University Press in association with Oxford University Press. Demy 8vo., pp. 215, with illustrations. Price: 11s. 6d. net.

The Medical Journal of Australia

SATURDAY, JANUARY 20, 1934.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

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THE TREATMENT OF CHOLECYSTITIS.

OMAR KHAYYAM was not alone in his plight when he wrote that in his frequent visits to doctor and saint he ever and anon came out by that same door wherein he went. In the world of medicine arguments are many and often futile. Nowhere is this more noticeable than in discussions on the medical and surgical treatment of certain conditions. For example, at any general discussion on the treatment of gastric ulcer physicians and surgeons will alternately advance their views, they will quote statistics to prove their theses, they will give details of cases in which treatment differing from their own has failed, they will perhaps admit grudgingly that there may be something in what the other side has to say, but they will quite often end by the triumphant statement: "This treatment has been successful in my hands and I intend to stick to it." The impartial listener, like the Persian poet, comes away not much wiser. Regarding the treatment of certain forms of cholecystitis much the same argument takes place.

There need be no question as to the type of treatment to be adopted in very mild cases of cholecystitis and in the very severe. At one end of the pathological series we have catarrhal and at the other gangrenous or empyematous cholecystitis. In text book classifications acute and chronic forms are recognized. Thus Graham, from the surgical side, recognizes these two forms only. Rolleston and McNee, however, describe catarrhal, acute and chronic varieties. We may conclude that catarrhal cholecystitis is a mild manifestation of the acute form. Catarrhal cholecystitis will be treated only by medical means, as will certain of the other mild acute forms. A gangrenous gall-bladder or one that is the seat of obviously advanced pathological change will be removed. When we come to the intervening types difference of opinion may be expressed. We do not propose to discuss in detail the treatment of cholecystitis; this will be done in the series of articles on treatment now being published in the journal. Mention will be made of certain results recently published in an endeavour to show that medical treatment can do a great deal for some patients, but that they are few in number and of a certain type.

J. M. Blackford, R. L. King and K. K. Sherwood presented to the annual meeting of the American Medical Association last June a paper in which they reported the condition of two hundred patients whose cholecystitis had been treated by medical means and who had been followed up for periods varying from five to fifteen years. Incidentally the following up of these patients is an achievement of note; it is much more usual to trace the history of patients after surgical operation than after purely medical treatment. The example is one that might be followed in Australian clinics. The study of these authors was based on a series of five hundred patients with cholecystitis who were not operated on. Those with local complications, however, such as cystic or common duct obstruction, cholangitis, pancreatitis, cholecystic empyema or associated pathological changes in the stomach, were excluded, as also were those who were operated on within six months of examination. The rest of the patients were divided into four groups: (i) those with indefinite symptoms attributed to cholecystitis; (ii) those with probable cholecystitis causing dyspepsia, with colic or atypical colic; (iii) those

with typical gall-bladder colic or typical gall-bladder dyspepsia or both; (iv) those with gall-bladder disease proved by X ray examination, or by stones found at operation for other troubles, or by a history of colic followed by jaundice. The first of these groups was discarded on account of the uncertainty of diagnosis. Of the remaining two hundred patients 37% had satisfactory relief over an average of more than two years without operation. Forty-eight *per centum* came to operation later or should have been operated on because of the continuation of symptoms. Only one patient died from gall-bladder disease, though 15% were dead. The treatment adopted by these authors consisted in the use of a suitable diet and in the taking of a saline aperient in the morning and bile salts before meals. Occasionally alkalis were prescribed. Infective foci were removed and physical exercise was ordered. The use of the duodenal tube for biliary drainage was not adopted.

In considering these figures it must be remembered first of all that, at least in the second group, the diagnosis may not always have been correct, and secondly, that the figures are percentages of a total that represents a select group. The 37% who had satisfactory relief become a much smaller group when the total, instead of being regarded as two hundred, is regarded as five hundred, as it should be. It is further to be noted that the average period of observation was only eight years. Disease of the gall-bladder often brings in its train some disorder of the pancreas, and gall-bladder conditions, even of the type included by these authors in their total of two hundred, are not infrequently followed by malignant disease involving the biliary tract; a period of eight years would probably be too short for either pancreatic or malignant disease to manifest itself. Turning to the other side of the picture, to make a proper comparison of methods it would be necessary to know the condition of a similar group of patients for periods of years after they had been submitted to operation. The freedom from symptoms would depend largely on whether operation was performed in the early or late stages of gall-bladder disease. The only possible conclusion is that figures obtained from a study such as that of Blackford and his co-workers do no more than point in a certain direction—they cannot be relied on as percentages. Admittedly, medical treatment, including drainage by the duodenal tube, not used by these workers, will suffice in mild infections; it may sometimes lead to the clearing up of a more severe cholecystitis; it may, of course, enable the patient to avoid operation if he is so determined. But a great responsibility is taken by the physician who undertakes purely medical treatment of a patient with a definite cholecystitis that does not yield at once to his therapy. Pathological change in the gall-bladder is generally progressive and involves the liver; the pancreas and its function become affected; the gall-bladder may be a local focus of infection; and malignant disease, even after years of apparent security, may supervene.

Current Comment.

ACUTE NEPHRITIS.

ACUTE nephritis is generally believed to be caused by the action of a toxin of some kind. Now that the focal sepsis theory has become widely accepted, septic foci are usually assumed to be either the source of the original toxin or the cause of prolongation of the disease. The focal sepsis theory is a comforting one if faith can be retained in it; for, providing sufficiently careful search is made, the physician is unfortunate if he is unable to find at least one possible septic focus. As a general rule a bee-line is made first of all for the tonsils, which are removed either because they are unhealthy or because they might be. It seems to be very difficult to decide whether a tonsil is healthy or not. At any rate, great numbers of them are removed nowadays, and probably quite rightly in many or most cases; but how many medical practitioners pause to consider what likelihood there is of benefit from tonsillectomy in any particular case? The shameful confession is that in the great majority of cases tonsillectomy as a means of removing a septic focus is merely a shot in the dark. Very little has been done in the way of comparing the histories of nephritics submitted to tonsillectomy with those of nephritics treated along general lines only. This and much more require to be done before a true evaluation of the measure can be made. In the meantime medical practitioners should preserve a judicial attitude and deal with each case on its merits, advising tonsillectomy when they see fit and not trusting blindly to lucky thrusts at possible sources of toxicity.

It is well known that acute nephritis frequently clears up spontaneously, leaving no apparent lesion; but in a certain percentage of cases, after the acute symptoms of œdema, hypertension, gross albuminuria and severe hæmaturia have subsided, small amounts of albumin and blood and occasional casts continue to be passed despite treatment. It is in these cases especially that septic areas as a source of toxicity are apt to be suspected. A. A. Osman, in the ninth of his series of papers entitled "Studies in Bright's Disease", has recently reported the results of his investigations into the treatment of this so-called "unresolved acute nephritis".[1] Osman employs this term because the term "subacute nephritis" implies that permanent structural changes have already taken place, or are in process of formation, in extent sufficient to encroach upon the kidney reserves. He states that he has had experience of a number of cases in which albuminuria persisted for some three or four years, but in which complete recovery eventually occurred. He points out that some years ago he conducted an investigation with the object of discovering the end-results of acute nephritis. He found that 70% of the patients in his series had completely recovered, though very few had been operated on for the removal of septic foci; the remaining patients all suffered from permanent renal damage.

In his recent investigation Osman observed the effects of tonsillectomy on seven patients. It is unfortunate that the numbers were so small. The patients varied in age from seven to twenty-six years. All had recovered from the acute signs and symptoms of nephritis, but had continued to pass small quantities of albumin and blood and had made no apparent progress towards recovery during a period of at least two months. Tonsillectomy was then performed. One patient recovered completely within a short time after operation; this was a girl, aged thirteen years, who had been in hospital for five months. It is interesting to note that her tonsils were apparently healthy to clinical examination and were seen to be mildly infected only when cut. The other patients remained *in statu quo ante*. One patient had had his tonsils removed before he came under observation; some small remnants were present, and there was empyema of the right maxillary antrum. The antrum infection was satisfactorily treated; but the tonsillar remnants were untouched. It scarcely seems fair to include this patient in the series; for, quite apart from the fact that he still had some tonsillar tissue, he had been ill for seven and a half years, and his blood reacted to the Wassermann test.

Osman points out that tonsillectomy is sometimes followed by acute nephritis. He quotes an illustrative case, in which the patient was still in hospital twelve months after operation.

The patients were observed for periods varying from five to twenty months after tonsillectomy had been performed. Osman considers a sufficiently long period of observation was allowed. He remarks that the results in this small series are in accordance with his clinical experience in a considerably larger number, and "strongly suggest that tonsillectomy is of very doubtful value in such cases as a means of hastening recovery".

The results of Osman's investigation are interesting and instructive. Unfortunately the evidence they provide is inconclusive. It is difficult to conceive how conclusive evidence could be obtained in a short time. It is probable that a true evaluation of tonsillectomy either as a prophylactic or curative measure will not be practicable until many years of experience have been obtained and many investigations have been made. Osman's observations are an encouragement for further research and a discouragement to a lazy faith in an unproven theory. Incidentally, it might be remembered that the tonsils do not constitute all the lymphoid tissue in the body.

[1] *Guy's Hospital Reports*, October, 1933.

UNDULANT FEVER.

In the past few years it has been found in the United States of America that undulant fever is not at all an uncommon disease. Three species of the causal organism are now recognized: *Brucella melitensis* (formerly *Micrococcus melitensis*), *Brucella abortus* (formerly *Bacillus abortus*), and *Brucella suis*. The treatment of undulant fever has been unsatisfactory. It is of interest therefore to read a recent report by I. Forest Huddleson and Howard W. Johnson.[1] These workers employ a substance named "Brucellin", which is prepared from separate broth cultures of the three species of brucella. After growth for sixty days at 37° C. the broth is clarified by centrifugation; the clear fluid is decanted from each of the three flasks, mixed, and filtered. After the usual tests for sterility it is ready for use. Before treatment is commenced the patient is tested for sensitivity by an intradermal injection of a soluble nucleo-protein fraction made from the three species of brucella. If there is no pronounced systemic reaction to the test, one cubic centimetre of "Brucellin" is injected intramuscularly; if there is a pronounced systemic reaction, 0·1 or 0·2 cubic centimetre only should be given at first. In all, four injections are usually given. In a series of eighty cases investigated by Huddleson and Johnson the response to treatment was rapid and satisfactory; in many there was no fever after the first injection. Not all of the patients with long-standing disease responded as quickly as those treated earlier; some required bigger doses and more injections. "Brucellin" causes a systemic reaction in many cases, but may be given without danger providing the medical practitioner is guided by the results of the test for sensitivity.

These workers appear to have discovered a specific remedy for undulant fever.

[1] *The American Journal of Tropical Medicine*, September, 1933.

Abstracts from Current Medical Literature.

PÆDIATRICS.

The Tuberculin Patch Test.

MAURICE GROZIN (*American Journal of Diseases of Children*, July, 1933) discusses the relative value and interpretation of the contact, non-traumatic methods of testing tuberculin sensitivity in the skin with the traumatic, with its cutaneous, intracutaneous and subcutaneous modifications. He refers to the greater precision of the Mantoux over the original von Pirquet technique, which is due to the possibility of introducing a quantitative factor and so increasing the sensitivity of the test. The disadvantages of the Mantoux test, enumerated by Grozin, are that it is painful and time-consuming when performed on a resisting child, and that occasionally even severe local and general reactions have followed. He states that for routine work a test is required which is reliable, painless and of simple technique, which consumes little time, is easy to interpret and is free from undesirable by-effects. Such is said to be the "patch" test, which Grozin had performed on 276 children. The method consists in applying to the skin a piece of adhesive plaster three centimetres square, on which one or two drops of old tuberculin have been placed. Distilled water is used as a control. The test is read after forty-eight hours; a reaction is shown by an area of erythema corresponding to the size of the plaster, studded with small confluent papules and vesicles, both visible and palpable. The location chosen is of no significance. To prevent a restless child from removing the plaster, it may be necessary to place a larger piece of this material over the first piece or to cover it with a bandage. The control patch may be red, but has no vesication, and disappears in a day, while the tuberculin reaction remains for several days. Patch test applications of Dick and Schick toxin have been found to produce no lesion.

Laryngeal Diphtheria.

O. F. TREGELLES (*British Journal of Children's Diseases*, June, 1933) describes the modernized form of aspiration treatment for laryngeal diphtheria in use at the North-Western Hospital. The procedure is to expose the larynx to view through a child's size Chevalier Jackson laryngoscope, in which the light bulbs are set in grooves in the side of the tube so that neither the field of vision nor the space for introduction of tubes is interfered with. The presence and extent of membrane are noted and a laryngeal culture is taken. An attempt is made to remove any loose or semi-detached membrane lying across or around the laryngeal aperture. The suction tube is then gently passed between the vocal cords at a moment when they are open; the child is then returned to the steam tent. If the obstruction is unrelieved, intubation is performed. An opinion may be formed from the laryngoscopic appearances as to whether a subsequent operation will be required; that is, when the false cords are so swollen that the true cords can scarcely be seen. If the first aspiration does not bring away much membrane, a second one from four to six hours after the administration of antitoxin will often remove large pieces. The respiratory difficulty sometimes following the use of intravenous serum renders this method inadvisable when aspiration is also practised. The author states that during the two years in which aspiration has been practised there has been a more sudden drop in the number of cases of laryngeal diphtheria requiring operation than could be accounted for by the general diminution of severity. At the North-Western Hospital, London, all patients admitted as suffering from croup, either simple or diphtheritic, are examined with a laryngoscope.

Loss of Weight in the New-Born.

IN an exhaustive analysis of the causes and nature of loss of weight in the first forty-eight hours of extra-uterine life, I. N. Kugelmass, R. Berggren and M. Cummings (*American Journal of Diseases of Children*, August, 1933) conclude that though universally sanctioned, this weight loss is unphysiological, and during the period of its operation the child's health is unduly vulnerable. They state that the neonatal growth gradient continues unaltered during the post-natal period, according to analysis of the transitional growth trends of the body as a whole, as well as of its tissues and organs. Post-natal loss of weight does not obtain in animals of any size other than human beings. The loss of weight is the result of dehydration and semi-starvation, conditions unfavourable for nutritional, physical and environmental adjustments benefiting the new-born. This loss can be prevented by the administration of a solution consisting of 6% gelatin (pH = 6·2), 3% dextrose and 0·5% sodium chloride at intervals of two hours throughout the first twenty-four hours after birth. The gelatin hydrates the blood and tissues, raises body heat and reduces clotting time. Dextrose brings the blood sugar content of the new-born to a normal level. Sodium chloride raises the initial low blood chloride content and favours hydration. The average loss of weight in infants thus treated was 1·7%, compared with an average loss of 7% in the controls. Birth shock, which causes weight loss, is therefore better combated by such a hydrating solution than by milk mixtures. There are less apathy, somnolence and stupor, and any acidosis is better compensated for. These studies showed also that male infants lost more than female infants, first-born more than their brothers and sisters, hospital babies more than babies delivered in private practice.

Poliomyelitis.

J. A. TOOMEY AND B. H. AUGUST (*American Journal of Diseases of Children*, August, 1933) have found a general correspondence between the harvest peak, especially for perishable crops, such as apples, and the epidemic peaks of poliomyelitis in Cleveland, United States of America, and suggest that such "perishables" may be one means of conveying infection. Such a conception, they say, would explain the somewhat dispersive and explosive type of spread of infection that occurs during the harvest peak for perishable fruits and vegetables. They claim to have discovered nearly similar findings in other countries, including Australia. The handling of infected fruit or vegetables by infected people, the eating of infected fruits *et cetera* by cattle, with subsequent infection of milk, and the spreading of infection from fruit to fruit by flies, could, it is stated, serve to explain the spread of the disease and heightened incidence at this time of the year.

Lactic Acid of Spinal Fluid in Meningitis.

A. G. DE SANCTIS, J. A. KILLIAN AND T. GARCIA (*American Journal of Diseases of Children*, August, 1933) have investigated the lactic acid content of the cerebro-spinal fluid in meningitis as a guide to diagnosis and prognosis. It had already been found by Killian that in meningitis the lactic acid of the spinal fluid was greater than that of the blood, but the sugar of the spinal fluid was less than that of the blood. The lactic acid content may fluctuate through a fairly wide range as compared with that of the sugar content. These authors have studied various types of meningitis, including the tuberculous variety, and confirmed these results. They accept 16 to 22 milligrammes per 100 cubic centimetres as the normal range for blood lactic acid, and about 78% of this for the spinal fluid. Forty-four patients in all were studied, daily analyses being made. Persistence in a high level of the lactic acid content, with a rise for 48 hours preceding death, was the rule in fatal cases. Within 24 hours following an improvement due to serum therapy the lactic acid content fell from 75 to 30 milligrammes per 100 cubic centimetres, and throughout the entire course of the observations the curves for sugar and lactic acid showed a reciprocal relationship. With an exacerbation of the infection there was observed a precipitate rise in lactic acid content and a fall in blood sugar content. In some cases of epilepsy, encephalitis, chorea and nephritic convulsions there is a rise in the lactic acid content of blood and spinal fluid. The authors state that a knowledge of the ratio between spinal

fluid lactic acid content and blood lactic acid content will be of considerable value in the diagnosis of inflammatory nervous diseases. The lactic acid content varies directly with the cell count; the results of glycolysis *in vitro* comprise the production of lactic acid by leucocytic activity.

ORTHOPÆDICS.

Parathyreoid Glands in Certain Skeletal Diseases.

E. L. COMPERE (*The Journal of Bone and Joint Surgery*, January, 1933) considers that there is no good evidence that Paget's disease is caused by hyperparathyreoidism and consequently that parathyreoidectomy is not a justifiable procedure in cases of ankylosing polyarthritis or Paget's disease unless there is more adequate evidence of parathyreoid gland involvement. He states, however, that an adenomatous tumour of one or more parathyreoid glands has always been found on *post mortem* examination and nearly always at operation. He considers that generalized *osteitis fibrosa* may be differentiated from other skeletal dystrophies which are clinically similar. The condition is characterized by a high serum calcium content, low plasma phosphates and increased excretion of calcium in the urine.

Arthritic Disturbances Associated with Parathyreoidism.

R. V. FUNSTEN (*The Journal of Bone and Joint Surgery*, January, 1933) discusses certain arthritic disturbances associated with hyperparathyreoidism. He finds this condition as a frequent concomitant of arthritis. It may be recognized by paroxysmal pain in the spine and abdomen and kyphosis which is due to partial collapse of the body of the vertebra owing to demineralization. Determinations of the calcium content of the blood and calcium excretion in the urine show as a rule an increase on treatment by means of cod liver oil and calcium gluconate, and will frequently result in improvement or arrest of the disease. Parathyreoidectomy, when there is an adenoma of a gland, results in almost immediate relief of pain and a feeling of relaxation in the joints.

Intermittent Claudication and Sympathetic Nerve Block.

F. L. REICHERT (*Annals of Surgery*, April, 1933) contends that relief of intermittent claudication in the arteriosclerotic person may be secured by interruption of the sympathetic pathways with alcohol. The usual tests to determine the efficiency of sympathetic interruption, such as the thermal response in the skin to spinal anæsthesia, to foreign protein or to peripheral nerve block, are not applicable in determining the effect of such interruption on the pain of claudication. Relief of pain is not infrequently obtainable, even in the absence of a rise in skin temperature. When the thermal response alone is the only criterion for or against sympathetic interruption, certain cases will be overlooked in which sympathetic block will be of distinct benefit. The author considers that a satisfactory diagnostic procedure to determine the relief from pain is a paravertebral injection of the sympathetic chain with "Novocain", which enables the ambulatory patient to assist the physician in judging whether or not permanent interruption will be beneficial. Twenty-five patients with intermittent claudication without gangrene have been materially and occasionally strikingly benefited by permanent interruption of the sympathetic chain by paravertebral injection of alcohol following the diagnostic use of "Novocain".

Arm-Chest Adhesions.

JACOB KULOWSKI (*Annals of Surgery*, May, 1933) discusses arm-chest adhesions. He considers the successful reconstruction or readjusting of the axillary space is the key to the relief of the condition. Certain inevitable and far-reaching sequelæ of axillary burn contracture make adequate reconstruction imperative, whenever possible. The simplest of these is the inability in some instances to wear ordinary clothes. The bones of the shoulder girdle become atrophic and their growth retarded from pressure of the scar and general disuse. Deformity of the ribs and limitation of thoracic excursion are present. Serious growth disturbances and scoliosis occur, particularly in children. Myogenetic and even myositic contractures may involve the axillary folds. Keloidal growths are not uncommon, and malignant degeneration has been observed. He states axiomatically that there is no short road to the reconstruction of arm-chest adhesions. The greatest danger lies in trying to do too much at one sitting. The work must be done in stages, and, further, it is a good plastic principle to remove all scar tissue before attempting any sort of reconstruction. He maintains that extensive undermining of large flaps frequently leads to necrosis and subsequent infection, and this may be even worse than the original condition.

Surgical Tuberculosis.

A. ROLLIER (*Surgery, Gynecology and Obstetrics*, August, 1933) considers that the use of large plaster apparatus in the treatment of surgical tuberculosis of bones and joints is physiologically and orthopædically wrong. Such apparatus keeps affected parts from contact with air and sun and produces softening of skin, atrophy of muscles, and decalcification of bones, the latter being due to the lack of light as well as the tuberculous process. He advocates the use of appliances that produce fixation and extension and at the same time provide for free exposure to air and sunshine. The bed is of special construction, moulded on large wheels with ball-bearings to prevent jarring. The mattress is firm and flat, and there is a sunshade for the head. In Pott's disease without deformity the patient simply lies on the hard mattress without pillows. If the patient is emaciated and the muscles atrophied, it is necessary to maintain the proper curvature of the spine, for which pillows filled with millet are used. Later a pillow filled with sand is used and the thickness gradually increased. Later, when the curvature becomes less and the skin less sensitive, a rectangular block of wood is used; the author states that this retains its shape and remains completely smooth and dry, thus preventing bed sores. When radiographs demonstrate vertebral consolidation the patient is encouraged to assume the ventral position, which, under heliotherapy, provides for restoration of the muscles of the back, which are the ideal and rational support of the vertebral column. In this position a small pillow under the thorax provides for lumbar lordosis, which is also assisted by the necessary movements of head and neck. The whole underlying principle is to avoid weight-bearing on the vertebræ. In hip disease he uses continuous plaster extension, heliotherapy, and an elastic band attached to a sandal to prevent foot-drop. Evacuation of pus is provided for by changes in posture. He has noted recalcification of the femur and acetabulum, reconstruction of the joint, and restoration of muscle function in 85% of cases. Recurrence more often follows in hip joints that have been ankylosed for some time than in hips that have been kept movable. Similar principles are followed in the treatment of disease in other joints. Heliotherapy causes disappearance of pain, infiltration and atrophy of skin, and recovery of muscles, followed by demarcation and progressive hardening of the foci, reorganization of sequestra, reformation of joint surfaces and even *restitutio ad integrum*. Untimely movements, active or passive, are always prone to produce relapses. On the contrary, heliotherapy may produce ankylosis when excision has been followed by pseudarthrosis. Cold abscesses are regarded as favourable reservoirs of antibodies which contribute in large measure to immunization; he deprecates their premature opening. Under heliotherapy even large abscesses become absorbed without any elevation of temperature. He considers that the appearance of a cold abscess is a good omen indicating the commencement of cure. Secondary infection changes the whole outlook and should be avoided at all costs; but if it occurs, adequate and early drainage must be provided. Sequestra may become absorbed or incorporated in the regenerating tissue, and should never be removed until demonstrably necrotic. He maintains an expectant attitude as long as nothing interferes with the spontaneous elimination of the sequestrum.

British Medical Association News.

SCIENTIFIC.

A MEETING OF THE VICTORIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at Warrnambool on July 22, 1933. Part of the report of this meeting was published in the issues of December 23 and 30, 1933.

Rest and Movement.

DR. FAY MACLURE read a paper entitled: "Rest and Movement" (see page 95).

DR. GERALD WEIGALL said that he was particularly interested in injuries to the *tendo Achillis*. Some years ago he had broken his own and was told by a surgeon who saw the injury that he should continue to walk on it. The surgeon who had made the first examination had not realized that the tendon was completely ruptured, and when the effusion subsided he was surprised to find that there was a complete separation of the divided ends. In the treatment of this injury Dr. Weigall had found that a raised heel on the affected side was a great help, but even with this he occasionally made a false step and ruptured the tendon again. This disability continued for about a year. He then fitted a leg-iron, hinged at the ankle, which prevented the ankle joint from being dorsiflexed to a right angle and which afforded complete protection against further injury to the damaged tendon.

DR. H. I. HOLMES referred to Dr. Maclure's description of the hoppling of the supraspinatus muscle, and asked whether Dr. Maclure carried out the surgical treatment before attempting to hopple the muscle.

DR. G. PENINGTON described the history of a patient who had suffered from monarticular arthritis of the hip bone. This patient illustrated the possibility of there being a psychological factor in joint injuries. The patient had developed an acute infective non-suppurative arthritis a few days after an operation for obstructed hernia. The arthritis spread from joint to joint, but subsided with medical treatment. There was some cartilaginous destruction in both knee joints and in the left ankle joint. Peripheral neuritis developed in both legs, but gradually improved until the patient was able to get about on crutches and later with sticks, but the pain became worse at the end of each day. She developed severe hyperalgesia of the sole of the left heel. The posterior tibial nerve was divided at the left ankle with relief, but after some months the nerve regenerated and the hyperalgesia returned. The pain prevented massage and active movement, and the patient developed a condition of spastic *talipes equinus*. As it seemed that there must be a functional element in the causation of this, she was treated by hypnosis by Dr. Norman Albiston and her symptoms were almost completely cured. This patient was not a neurotic person, but had suffered very severe pain for four years. She was now able to work. She still had her arthritis, but very much improved by the hypnotic suggestion.

DR. THOMAS KING said that he was extremely interested in Dr. Maclure's paper and appreciated his highly original handling of the subject. In discussing the importance of rest to relieve pain in an injury he indicated the use of painless passive movements, with the object thereby of obtaining a movable joint. Admitting that any method handled by a master gave good results, none the less it was now well recognized that passive movement in recent joint injuries was harmful in most people's hands. The late Hamilton Russell formulated the following laws forty-five years ago, and they were almost universally accepted orthopædic principles of the present day.

1. In fracture involving or in the immediate vicinity of a joint, where there is no gross displacement of the articular surfaces, stiffness of the joint never supervenes, except as a result of passive movement carried out by the surgeon.

2. In fractures involving joints in which there is some displacement or irregularity of the joint surfaces and in which, therefore, some impairment is inevitable, such stiffness will be greatly aggravated by the employment of passive movement; and complete abstention from passive movement by the surgeon will yield the best result obtainable. (Nowadays a perfect anatomical reposition of the fragments would be required.)

3. When great stiffness is present, in spite of the surgeon's efforts to increase the mobility by passive movements, such stiffness will persist so long as the surgeon persists.

4. When the surgeon abandons the case in despair, improvement will at once commence.

Condensing these four laws into one weighty generalization, they might say that in fractures involving joints the most potent cause of stiffness was the so-called passive movement performed by the surgeon. Immobilization was never a cause of stiffness. The maximum degree of permanent usefulness was to be obtained by immobilization alone (*Intercolonial Medical Journal*, August 20, 1899).

In regard to the treatment of a sprained ankle, Dr. King followed the same principle, so aptly described by Dr. Maclure as "hoppling", but he effected a similar object in a somewhat different manner. The hæmatoma about the anterior attachment of the external lateral ligament was injected with 2% "Novocain" solution and then pressed away by firm massage. A heavy Unna's paste cast was then applied from the toe clefts to the tibial tuberosities. The patient was encouraged to walk immediately or within one or two days. On beginning to walk each morning after the night's rest, the ankle was stiff and painful, and walking seemed almost impossible, but after about half an hour the pain gradually wore off, if walking was persisted with. Subsequent massage *et cetera* were superfluous. In regard to "tennis elbow", he had found, like Dr. Maclure, that it occurred in all manner of sports and crafts, especially among wood-choppers. In all cases the tender point was constantly just below the external epicondyle of the humerus. It appeared that some tearing of a few fibres of the common extensor tendinous origin was the lesion, though there was no agreement about this. Whilst the following method (Mills) was not always successful, dramatic results were seen in over half the cases, provided the manipulation had been successfully carried out. One met quite a sufficient number of people who had been almost instantaneously cured by osteopaths of their "tennis elbow" to be convinced that a successful manipulation of this injury could be an extremely successful surgical procedure, which every medical practitioner should be acquainted with. The patient should be seated in a chair and engaged with pleasant conversation. The operator placed his thumb firmly over the tender site and rested the lower end of the humerus on the injured side, over his bent knee (an efficient fulcrum for all manipulative procedures). Then the wrist was hypervolar flexed and the forearm fully pronated. Now at this, the crucial, stage, with the patient engrossed in the conversation and preferably talking about himself, the elbow was suddenly fully extended. Unless a sudden crack was heard and the elbow could now be more fully extended than before, success would not be met with. As a rule, after the initial pain had worn off the arm was almost completely free from pain, which entirely disappeared within a few days.

DR. A. E. COATES expressed his appreciation of Dr. Maclure's paper. With other members, he had felt he was sitting at the feet of a master. Dr. Coates asked if Dr. Maclure considered that cases of so-called "painful shoulder" were all due to tendonitis of the supraspinatus tendon, and suggested that some of them were due to some less easily diagnosed cause.

DR. J. KENNEDY expressed his gratitude to Dr. Maclure and said that supraspinatus tendon injuries were fairly common in industrial insurance work. He (Dr. Kennedy) had not seen any very good results from treatment short of operative interference. With regard to his experience

of manipulative treatment in out-patient departments, Dr. Kennedy said that the results were not so good as when a patient could be put to bed for a week at least at complete rest under expert observation after manipulation had been carried out.

Dr. Maclure, in reply, expressed his thanks for the interest of members and for the reception of his paper. He referred to the great difficulty in diagnosing and treating sacro-iliac pain, and he sometimes hoped that patients suffering from this condition would seek other advice. Dr. Weigall's suggestion for the hopple of a damaged *tendo Achillis* was a very useful one, particularly as it had given good results in his own personal experience. He was most interested in Dr. Penington's description of the patient who was relieved by hypnotic suggestion. It was true that many hysterical patients referred their troubles to joints. It was interesting to recall that Hamilton Russell many years before had addressed the first meeting of the Surgical Association in Melbourne and had given as the title of his address "The Psychology of the Knee Joint". Regarding the condition of "painful shoulder", Dr. Maclure had a patient recently complaining of this condition, who was suffering so much inconvenience and pain that she demanded that something should be done. X ray examination showed opacity in the region of the tendon of the supraspinatous muscle. Exploratory needling of this area gave no relief, but the operation of splitting the insertion of the supraspinatous tendon revealed a little nest of calcium soap, the removal of which gave the patient relief. Dr. Maclure said that before operation or manipulation for this condition modified rest and hoppling should be tried for about a fortnight.

A MEETING OF THE VICTORIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at Wangaratta on October 21, 1933.

Intracapsular Fractures of the Neck of the Femur.

DR. THOMAS KING read a paper entitled: "Recent Intracapsular Fractures of the Neck of the Femur: A Critical Consideration of Their Treatment and a Description of a New Technique." This paper was published in the issue of January 6, 1934. This account of the meeting was unfortunately omitted from that issue.

DR. VICTOR HURLEY offered his congratulations on the excellent presentation of the technique and treatment of a very difficult surgical problem. Dr. Hurley said that he was convinced that the method would prove to be the most satisfactory for treating fractured neck of the femur. It seemed to obviate the essential disadvantages of all previous methods. Using Whitman's method of impacting a fibular bone graft into the head of the femur, Dr. Hurley had had good results in two out of three cases, but he considered that Dr. King's method was a definite advance on Whitman's, and also on the Smith-Peterson method, of which it was a modification. He admired Dr. King's skill and patience in developing all the details of the technique and his ingenuity in carrying it to success. One disadvantage of the method was that it was a highly specialized procedure, only possible in a well equipped hospital with specially trained staff and special apparatus, including a shock-proof portable X ray set, but possibly in time methods might be evolved to enable the method to be carried out with less elaborate apparatus and so make it more generally applicable.

DR. HUGH TRUMBLE heartily supported Dr. Victor Hurley in his admiration of Dr. King's great thoughtfulness and high degree of mechanical ability. One possible defect of the method was that it would not be available to surgeons of less ability and with less efficient apparatus. There was no doubt that Dr. King had improved on all existing methods. One of the great difficulties in the past in treating fractured neck of the femur had been the danger of infection. With the usual open operation incisions were large and the operation tended to be bloody and rather lengthy, and infection often occurred even in the hands of competent surgeons. Dr. King's relatively "closed" method should largely obviate the danger of infection.

Dr. Trumble also demonstrated a walking-iron, consisting of a padded seat to fit both ischial tuberosities, and having a supporting pad to fit anteriorly against the pubis. This support was intended to replace the various types of walking caliper which were notoriously unsatisfactory and uncomfortable.

Dr. King, in reply, thanked Dr. Hurley and Dr. Trumble for their appreciation, and said that the common occurrence of infection after operation for fractured neck of the femur had been the chief reason for setting out to devise the new method which he had presented.

NOMINATIONS AND ELECTIONS.

THE undermentioned has been nominated for election as a member of the New South Wales Branch of the British Medical Association:

Pollock, James Lesslie, M.B., B.S., 1933 (Univ. Sydney), 1, Melnotte Avenue, Roseville.

The undermentioned have been elected members of the New South Wales Branch of the British Medical Association:

Burns, Colin Michael, M.B., B.S., 1930 (Univ. Sydney), 27, Fort Street, Petersham.

Birchall, Ida Lois, M.B., B.S., 1933 (Univ. Sydney), c.o. Dr. H. Thomas, Manly.

McNaught, Ian Webster, M.B., B.S., 1931 (Univ. Sydney), 2, Audley Street, Petersham.

Short, D'Arcy Nelson, M.B., B.S., 1931 (Univ. Sydney), Abuklea Road, Epping.

Spencer, Stanley Livingstone, M.B., B.S., 1930 (Univ. Sydney), 46, Archer Street, Chatswood.

Post-Graduate Work.

POST-GRADUATE LECTURES IN MELBOURNE.

THE Melbourne Permanent Post-Graduate Committee announces that arrangements have been made with Professor David Barr, Professor of Medicine, Washington University, and Physician-in-Chief, Barnes Hospital, St. Louis, United States of America, to deliver a course of six lectures in Melbourne towards the end of July, 1934.

The subjects of the lectures will be as follows:

1. The functions of the anterior lobe of the hypophysis.
2. The suprarenal glands and Addison's disease.
3. Thyreotoxicosis.
4. Hyperinsulinism and related conditions.
5. The relation of the parathyreoid glands to calcium metabolism.
6. Epidemic encephalitis.

Further details in connexion with this course of lectures will be announced at a later date.

Correspondence.

THE HEALTH OF THE PEOPLE OF PAPUA.

SIR: I would venture to direct your attention to a letter by Sir James W. Barrett, published on pages 835-6 of THE MEDICAL JOURNAL OF AUSTRALIA under date December 16, by reason of its being a criticism of the Government of the Commonwealth in the matter of sending Papuans for medical training to the School of Tropical Medicine at the University of Sydney.

Sir James Barrett would seem to consider that Fiji would have been a better place to have sent them. At one

time this was my own view, but when I found that all the authorities were only too pleased to welcome the Papuans at the School of Tropical Medicine, I at once saw that it was the natural place for them to go to. Papua is a territory of Australia. The Commonwealth Government has already established a School of Tropical Medicine there. Many, at least, of the teachers of the school have had tropical experience, and the school is in charge of highly competent teachers who have the advantage of working in close collaboration with a great and well established university, with all resources for giving a first class medical training, a better training than, I think, could be given at a place like Fiji. It would seem more natural for the natives of an Australian territory to be trained in an Australian university rather than elsewhere.

I have every respect for the work done in Fiji. However, I note Sir James Barrett's reference to "anyone who will read their medical journal", *The Native Practitioner*. I quite agree that if this journal were entirely run by the native practitioners of Fiji their journal would be strong evidence of high attainments; but I cannot believe it is so run. A few months ago three numbers of their journal came into my hands, full of well written articles *et cetera*. However, among the articles I saw an article written by a Fijian, who I knew had worked in Papua for one of the missions. In fact, the article was with reference to his work in Papua and was written in excellent English on his return to Fiji. This Fijian was duly introduced to me on his arrival in Papua, and I was given to understand that as he spoke neither English nor Motuan it would be impossible for me to discuss matters with him. The article as printed contained misstatements and would imply that the Fijian had taken a far more important share of the medical work in Papua than he had in point of fact. I have not the article by me now, but I remember the undermentioned as having occurred in same.

In the article which was purported to be signed by the Fijian it was stated that on his arrival in Papua there were no government native hospitals in Papua. This is quite contrary to the fact. There were government hospitals many years before the Fijian ever came to Papua. I can remember a native hospital at Samarai under a medical officer, Dr. Jones, in 1908. I can also remember a native hospital at Port Moresby in 1910 under a medical officer, Dr. Goldsmith. A reference to the estimates of Papua and of British New Guinea would show that money was put on the estimates for native hospitals even before this. Hence the article as printed contained a direct misstatement of fact. Perhaps the editor of the article knew little or nothing of Papua and did not entirely understand the Fijian.

The article as printed also stated that when the Fijian left there were government native hospitals at both Port Moresby and Samarai. This, of course, is quite correct, but, combined with the previous incorrect statement that there were no government hospitals on the arrival of the Fijian, it would lead a reader to assume that the arrival of the Fijian in Papua has in some way stimulated the government to take up native hospital work. There is not, however, the slightest atom of truth in any such suggestion.

Again, the Fijian, as printed, recounts in the article the work which was done at the mission station while he was working for the mission. This would naturally lead a reader to assume that the Fijian was in charge of such work, but in point of fact the Papuan Government at the time was subsidizing a qualified doctor (as well as subsidizing the Fijian) to be in charge of the medical work of the mission, and the Fijian was working under this doctor.

Sir James Barrett, at the end of the last paragraph but two of his letter, states that his criticism is based, not on opinion, but on the facts of the case; but does he know the facts? I venture to suggest that he knows very little of the facts of the education which the Papuans have received in Papua and at Sydney. I have never heard of his being in Papua, nor have I seen or heard of his visiting the School of Tropical Medicine since the Papuan students first came there. Had he lived in Papua during the last thirty years I have worked there, I could hardly have missed hearing of his visit, and had he visited the Sydney University during the last few months the Papuans have been working there, I must have known.

I regret having had to write in the argumentative strain I have had to. I regret because I realize that Sir James Barrett has the welfare of natives and their medical training at heart, and in this I am quite with him, but I differ from him in that he would prefer that their training should be given at Suva rather than at Sydney University.

Whilst it is difficult to see what bearing the first few lines of his letter have on the training of Papuans, I feel I must make a few remarks on the same. It is true that there are some 300,000 natives in the Territory of Papua, probably, I suppose, rather more than this in the Territory of New Guinea, and perhaps half a million in Dutch New Guinea. Doubtlessly nearly all those living on the coastal flats of New Guinea are infected with malaria, but this does not necessarily mean anything very serious. I was certainly infected with malaria in 1904, and suppose that since then I have always been, and am now, infected with malarial parasites hidden away somewhere in the body, but, nevertheless, I have never lost the whole of a day's work on account of such malaria. Again, Australia has been in charge of the Territory of Papua for some twenty-six years. I do not know how many officers have retired, pensioned, on the ground of ill health. I do not think very many, and of those who have so retired I think only one or two (if any) will have retired on the ground of malaria.

The present course which the Papuans are taking at Sydney University is only a beginning. I venture to hope a beginning in that more Papuans will visit Sydney and that some of the Papuans will come again and be educated to even higher things. But the progress they have made assures me that Sydney University need not fear comparison in any way with the Medical School at Fiji.

Yours, etc.,

W. M. STRONG,
Chief Medical Officer, Territory of Papua.

School of Public Health and Tropical Medicine,
The University of Sydney,
December 21, 1933.

LEAD POISONING IN INDUSTRY.

SIR: The references to lead poisoning at Mount Isa Mines, Queensland, in your issue of December 16, 1933, would tend to give an entirely erroneous idea of the true state of affairs, and in fairness to the management, I would request that the following details be published.

It is unfortunately true that no provision was made for, or thought given to, the control of lead poisoning when the plant was designed and built. The fault here lies in the non-cooperation of the engineering and industrial medical aspects, a matter which urgently requires world-wide recognition and adoption.

When production commenced at Mount Isa in June, 1931, measures along all approved lines were undertaken. Mount Isa Mines, Limited, prosecuted an active, detailed and costly campaign, which at the time of the Duhig-Watson visit was just succeeding in its object.

I would like also to state that every mechanical operating and medical avenue of control suggested in the Duhig-Watson report and within the company's powers was in operation or process of installation at the time of the visit.

A peculiar set of circumstances of an industrial and medical nature alone prevented the full realization of the ideals long held by the management, who were completely cognizant of the problem, and the urgency and completeness of the measures necessary for its solution.

The one very real benefit to Mount Isa and to Queensland has been the recognition of a necessary and careful

standard of diagnosis and a more comprehensive *modus operandi* of certification in cases of compensatable lead intoxication.

Yours, etc.,

R. H. VON DER BORCH,
Chief Medical Officer, Mount Isa Mines, Limited.

Mount Isa,
Queensland,
December 26, 1933.

THE PRE-SCHOOL CHILD.

SIR: I am surprised to read in your leading article of December 23 that "there is no organization, no branch of a health department, charged with the special care of the pre-school child".

I cannot speak for the other States, but this is certainly not true of Queensland. On the contrary, the care of the pre-school child is as much part of the duty of our Child Welfare Department as the care of the infant. To the best of our ability, and as far as the means granted to us will allow, we are endeavouring to educate mothers in the importance of avoiding malnutrition and other evils which may follow from deficient diets, in the formation and preservation of sound teeth, in the prevention of infections (a peculiarly difficult problem), and in other matters which concern the health of the pre-school child.

We have not succeeded in attracting as many young children to our centres as we have of the infants, but the numbers that come are increasing. Every year a Child Welfare car tours the railways of the State. Newspaper articles, booklets and leaflets have been continually issued and distributed. I need hardly say that these are not concerned with the treatment, but with the prevention of disease.

Yours, etc.,

A. JEFFERIS TURNER, M.D., D.T.H.

Infant and Child Welfare
(Director's Office),
Wickham Terrace,
Brisbane,
December 26, 1933.

WORKERS' COMPENSATION PRACTICE IN NEW SOUTH WALES.

SIR: Schedule D has come to an end, and now we can read Section 10, Subsection (4) (*a*), carefully for ourselves and revalue its simple provisions and reinterpret its simple words. We face the future with more experience in workers' injuries, and we have the benefit of the experience of Dr. R. M. Mackay, Chief Medical Referee of the Commission.

Schedule D was an evasion of Section 10. It was full of faults and had the hall-mark of a temporary expedient. It is a wonder we put up with it so long. Apparently our Association rushed into this compromise with the associated insurers so that we could have a ready means of collecting our fees.

Dr. Mackay, in his interesting address, hinted that workers' compensation today was too great a burden on industry and that unless we continued to evade Section 10 more and more, Parliament might introduce an amending act that might be worse for us than the present Act.

First, it is by no means agreed that industry cannot carry its compensation burden, and at any rate that is not our business. All we can say is that we carry our share and find it no burden.

Secondly, we are not certain that such an amending act is likely to be brought forward; and, even so, it may possibly be more beneficial than otherwise to us. We would have our share in formulating such an amendment, and it would be a great opportunity for us to seek powers to sue for fees from the employer or his insurers and to have some standing to urge our claims before the Commission. Perhaps after six years we can now see that in 1927 it would have been wiser for us to have fought for these principles than to have taken the easy road to Schedule D.

Thirdly, it is common experience that it is best to leave law-making to Parliament and not to tamper with its acts.

Dr. Mackay also propounds a fallacious theorem when he draws comparisons between the worker's wages and the cost of his treatment, and deduces from this that therefore the medical costs are too high. The Act is between master and worker; the master is clearly liable for the worker's treatment at the usual rates the former would have to pay himself; therefore, before logical deductions regarding relative costs can be made, the fees for treating the worker must be compared, not with his own, but with his master's earning power.

When a doctor attends a worker (within the meaning of the Act) he really attends the servant at his master's request and expense, and, long before any compensation act was evolved, the charge for attending a servant was, by convention, the same as that for the master, if the attendance was at the latter's request and in the absence of any express agreement.

Dr. A. M. Davidson and Dr. Richard D. Davey, in their letters of the last issue of the journal, express irrefutable views which most of us have come to realize.

In each locality there are "legal" fees governed by custom or practice. These vary from place to place, as the Act recognizes. We are either worth these fees or we are not worth them. If we are worth them, then we should unflinchingly demand them; if our services are not worth the regular rate, let us say so and take less; but do not let us underrate our services by scheduled reductions which are ridiculous from a business standpoint and humiliating to our profession.

Yours, etc.,

G. H. APPEL.

Moruya,
New South Wales,
December 31, 1933.

APPENDICITIS AND ITS TREATMENT.

SIR: Dr. Pomroy's paper on the above, in THE MEDICAL JOURNAL OF AUSTRALIA of December 30, 1933, is one of the most important which have appeared in the journal. Many lives have been lost by ignoring the principle so convincingly advocated by him, that while every patient suffering from appendicitis should be operated on within the first forty-eight hours, if possible, operation during the next three or four days is more dangerous than watchful medical treatment. It is earnestly to be hoped that the profession will carefully consider the facts brought forward by Dr. Pomroy.

While personally endorsing the position he has taken up, may I venture to make one or two criticisms. It is not correct that the turbid fluid present in large quantity in many cases of acute appendicitis is always sterile; it is in any case a culture medium and should always be drained away. I could furnish many examples of the wisdom of this advice.

I have met with several instances of an appendiceal abscess bursting into the bowel, with recovery of the patient, but I do not believe that a definite collection of pus is ever absorbed. I have seen double pus tubes in an old lady of seventy, the pus being the consistence of soft cheese and the history pointing to the infection having occurred thirty years previously. Operation was called for at this late stage because of acute symptoms due to torsion of one of the tubes.

When operating for appendiceal abscess it is better to remove the appendix if it can be done easily, without breaking down the limiting adhesions. A transverse incision is best for such a condition, cutting through skin and aponeurosis and separating the fibres of the internal oblique and transversalis.

Opening a pelvic abscess through the rectum often cannot be avoided in a man, but is highly improper in a woman. The vagina is a natural drainage tube; the pus lies in contact with its upper part; then why not incise there?

The drainage tube should be short and on no account should extend to the vulva, where it is exposed to noxious influences. It is wise to split the tube so as to minimize the pressure on inflamed viscera. A so-called "dressed tube" is foolishness; gauze in a tube quickly becomes saturated and then acts as a cork, blocking drainage.

The best time to operate is not three months after subsidence of the acute symptoms, but three weeks after; because, as Treves taught, an immunity has been established, but this does not last indefinitely.

Forty-eight hours is too soon to move the bowels. Anyone who has watched a rectal saline given while the abdomen is open and noted how the fluid will rapidly make its way as far as the caecum, will agree with me in this.

Finally, a suprapubic drainage tube should be avoided whenever possible; it is right in the small intestine area and tends to give rise to crippling adhesions and intestinal obstruction. The plan which I have several times advocated is far better. Pass two fingers of the left hand through the wound, the finger tips pointing to the right. Feel for the pulsation of the deep epigastric artery. Make a one-inch transverse incision through skin and aponeurosis just external to the vessel. Pass a Howard Kelly forceps between the fibres of the muscles and through the peritoneum. Take hold of the end of the split rubber drainage tube. Pull it through the stab wound. Pass the other end to the bottom of the pelvis, keeping close to the bony wall of the pelvis in its course, that is, as far away from the small intestine area as possible. Cut the tube almost flush with the skin and cover the opening with squares of sterile gauze. Change these as frequently as they become wet. A nurse sitting by the patient with a sterile forceps in a bowl of spirit can do this with little trouble and without disturbing the main dressing. Rotate the tube gently in twelve hours and remove it directly it ceases to drain, which is generally in twenty-four or thirty-six hours.

Yours, etc.,

233, Macquarie Street, Sydney,
January 2, 1934.

RALPH WORRALL.

Books Received.

CATECHISM SERIES: ANATOMY (ABDOMEN), Part IV, Fourth Edition, by C. R. Whittaker, F.R.C.S.E., F.R.S.E.; 1933. Edinburgh: E. and S. Livingstone. Crown 8vo., pp. 84. Price: 1s. 6d. net.

HANDBOOK OF MIDWIFERY FOR C.M.B. STUDENTS, by W. O. Greenwood, M.D., B.S., F.R.S.E.; 1933. London: John Bale, Sons and Danielsson, Limited. Crown 8vo., pp. 120, with illustrations. Price: 5s. net.

NATURE AND NURTURE, by L. Hogben, M.A., D.Sc.; 1933. London: Williams and Norgate, Limited. Demy 8vo., pp. 144. Price: 6s. 6d. net.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser", pages xvi, xvii

DISTRICT HOSPITAL, KALGOORLIE, WESTERN AUSTRALIA: Resident Medical Officer.

LAUNCESTON PUBLIC HOSPITAL, LAUNCESTON, TASMANIA: Resident Medical Officer.

RENWICK HOSPITAL FOR INFANTS, SYDNEY, NEW SOUTH WALES: Honorary Physicians.

SAINT VINCENT'S HOSPITAL, MELBOURNE, VICTORIA: Honorary Officers.

THE ADELAIDE CHILDREN'S HOSPITAL, ADELAIDE, SOUTH AUSTRALIA: Resident Medical Officers.

THE QUEEN'S (MATERNITY) HOME, ROSE PARK, ADELAIDE, SOUTH AUSTRALIA: Resident House Surgeon.

THE TOWNSVILLE HOSPITALS' BOARD, TOWNSVILLE, QUEENSLAND: Junior Resident Medical Officer.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Brisbane Associated Friendly Societies' Medical Institute. Chillagoe Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL are advised, in their own interests, to submit a copy of their agreement to the Council before signing. Lower Burdekin District Hospital, Ayr.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	Combined Friendly Societies, Clarendon and Kangarilla districts. All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 205, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

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